BRAIN.

PART IV., 1898.

Original Articles and Clinical Cases.

PATHOLOGY OF A CASE OF FRIEDREICH'S DISEASE.

With a Summary of previously reported cases.

BY H. MACKAY, M.D.

THE case of Friedreich's disease, the morbid anatomy of which I here report, was one of a family described clinically in the *American Journal of the Medical Sciences*, vol. cviii., 1894, p. 151.

To save the trouble of reference, I append here a brief note of the clinical condition of the patient during life.

H. W., male, unmarried, aged 26 at the time of examination, was one of a family, three members of which, *i.e.*, two sisters and himself, have been under my observation for Friedreich's disease. A fourth member, a sister, had already died from the same complaint. There is no hereditary transmission of the disease, but various spinal neuroses are present in collateral relatives of the patient, and a second cousin probably died of the same disease.

The patient suffered from measles and scarlet fever in his sixth year, soon after which weakness of the legs and staggering appeared. At twelve he was unable to move about; at fourteen the arms began to be attacked, and by his twenty-second year these limbs were helpless.

He came under my notice about this time and presented a typical picture of the later stage of the disease. His power of voluntary movement was limited to slight motion at the joints of the upper limbs, raising and rotation of the head, and some control over the muscles of expression. He was also able to roll

over in bed, and to raise the trunk on the elbows when supine. His deep and superficial reflexes were abolished, with the exception of normal pupillary reactions to light and accommodation. His sensation was everywhere well preserved, and his special senses acute.

Coördination had been much impaired before the limbs became fixed. He had lateral nystagmus and frequent twitchings in the facial muscles. His speech was laboured and explosive. His intelligence was good, and he had complete control over bladder and bowel. The spine showed a lateral curvature. The optic discs were normal.

The patient suffered from asthma, and from cardiac enfeeblement, but without recognisable dilatation or valvular disease.

In the winter of 1897, when 30 years of age, he contracted measles for the second time. Symptoms of collapse speedily supervened, with cyanosis and heart failure from which he died.

The autopsy, some twenty-eight hours after death, showed the following. Permission to open the chest was not obtained.

Cranium.—Much adhesion of Pacchionian bodies, otherwise no abnormality.

Membranes.—Normal; no excess of fluid in subdural space.

Brain.—To naked-eye inspection normal. The grey and white matter were natural on section, and the convolutions well marked.

Cerebellum.—Well developed and of average size. Measurements in the three chief planes:—11·8 c.m. × 5·3 c.m. × (between vermiform processes) 4 c.m. Schultze gives, as average figures, 11·5 c.m. to 12·5 c.m. × 5·25 c.m. to 7·5 c.m. × 3 c.m. to 4 c.m.

Medulla.—Visibly smaller than normal. Vagi, hypoglossi, and other cranial nerves appear to be normal.

Cord.—The dural sac is loose and baggy, and the contained fluid excessive in amount. The latter looks like normal cerebro-spinal fluid. The dura appears thickened, but the pia has no abnormal appearance. The cord is evidently shrunken in size. On section the posterior column is everywhere greyish and translucent, and the same appearance is visible in the posterior part of the lateral columns. The posterior spinal roots are visibly shrunken in size. The anterior appear normal.

The contents of the cranium, and the spinal cord, were placed in Müller's fluid, together with portions of the following peripheral nerves: the sciatic and the anterior crural of the right side, the anterior tibial and internal plantar of the left side. From the upper limbs were taken portions of the right

median and ulnar, and of the left radial and posterior interosseous. These latter were hardened in Erlitzky's fluid followed by methylated spirit.

For defining the areas of degeneration in the cord and medulla, preference was given to Schäfer's modification of the Pal-Weigert method. By this process, myelin-bearing fibres, whether healthy or degenerating, stand out in black or blue-black against the white ground of sclerosed or non-medullated tissue. For differentiating healthy from degenerate myelin, Hamilton's modification of Marchi's process was used. For investigating the neuroglia, to which, since the appearance of Déjérine and Letulle's observations [8] considerable interest has attached, attempts were made to use Weigert's—now classical —1895 method,[33] but owing to the hardening of the material in Müller's fluid, the attempt was not successful. Mallory's 1895 method [34] failed also for the same reason. Better results were obtained by a preliminary stain in Van Gieson's fluid, followed by Mallory's phospho-molybdic or phospho-tungstic hæmatoxylin.[35] By this means an excellent triple stain is obtained, in which the dark axis-cylinders and glia cells are well distinguished from medullary sheaths (yellow) and connective tissue other than glial (red-violet). For the peripheral nerves Schäfer's method answered admirably, while for axis-cylinders aniline blue-black, benzopurpurin, or Mallory's hæmatoxylin was used. For the spinal ganglia toluidin-blue and eosin, hæmatoxylin and eosin, or picro-carmine were employed.

The actual changes found were as follows :—

Cord.

T. S. Lower Lumbar (L. V. and IV.)

Posterior Column.—There is well marked sclerosis, most marked in the posterior part of the column. At the level of the 5th lumbar this involves the posterior third of the column. The anterior third, next to the commissure, shows a large number of well-preserved fibres, but also a few swollen and degenerate ones. The middle third of the column shows a gradual transition between the conditions described. In the posterior third very few stained fibres can be seen, and these are scattered at wide intervals throughout the sclerosed area. A band of comparatively well-preserved fibres lies along the mesial side of the posterior horn on each side, separating the sclerosed portion from the grey matter. This, which is continuous with the well-pre-

served fibres lying dorsal to the commissure, evidently represents Marie's cornu-commissural tract.

Of the posterior root fibres, those entering the gelatinous substance (middle group of Lenhossek), those entering Burdach's column (mesial group), and those occupying Lissauer's border-zone are all intensely degenerated, the course of individual fibres passing horizontally being marked generally by isolated droplets of myelin, while those transversely divided display swollen sheaths and an absence of axis-cylinders.

The sclerosed area includes the septo-marginal tract of Bruce and Muir and the central oval tract of Flechsig, neither of which contain healthy fibres.

At the level of L. IV. the dark stained fibres occupy only the anterior fourth of the posterior column. The remaining three-fourths are degenerated, the posterior portion the most severely. About the centre of the posterior column, however, a small, linear tract of fairly well-preserved fibres appears, bordering the posterior fissure. This has not the oval shape of Flechsig's *centrum ovale*, and probably corresponds to a portion of Bruce's septo-marginal tract.

Lateral Column.—At the lower level, L. V., there is apparent under the low power a lightening of the myelin stain in a triangular area occupying the posterior part of the column, but separated from the posterior horn by the well-preserved fibres of the lateral limiting layer. At the higher level, L. IV., the degeneration is more pronounced, and corresponds with the position of the crossed pyramidal tract. In connection with Marie's view[36] that the degenerated area in the lateral column does not represent the crossed pyramidal tract, it was noted that the degeneration affected equally the large and the small fibres of the area. It is equally true, however, that many large fibres remained unaffected throughout the tract. As will be seen afterwards, the size of the lateral sclerosed area diminished from the mid-dorsal region upwards, but it increased from the lumbar to the mid-dorsal region, and at the latter level well-marked degeneration appeared in the fibres of the direct pyramidal tract, which might account for the lateral diminution from that level upwards. There is distinct neuroglial overgrowth present, with an increase in the number of round, granular glial nuclei.

Anterior Column.—Normal.

Grey Matter. Posterior Horn.—There is extensive degeneration both of the vertical (ascending) and of the transverse (entering) fibres in the caput cornu.

FIG. 1.

FIG. 2.

There is no trace of any healthy nerve cells, while the adjoining large cells of the intermediate grey substance are well preserved. The reticulum of fine medullate fibres in the substance of the horn is fairly but not well, preserved.

Anterior Horn.—The large motor cells with their processes, and the fibrillar plexus are well preserved.

The Central Canal is occluded by a mass of small, round cells, which take on nuclear stains deeply. No trace of ependymal epithelium remains.

The posterior roots, spinal ganglia, and anterior roots will be described in detail later. It is sufficient here to say that the posterior roots were intensely sclerosed.

Upper Lumbar and Lowest Dorsal L. I. to D. XII.

Posterior Column.—At this level the postero-median fissure is for the first time recognisable. Previously—at lower levels—it had only been obscurely suggested, and had contained no prolongation from the pia. There is as yet no differentiation of Goll's from Burdach's column.

The area of stained medullate tissue in the P.C. is now limited to the tract of cornu-commissural fibres, which fill the apex of the posterior column, and occupy its lateral margins close to the grey matter. This area, however, contains a considerable number of swollen and otherwise degenerate fibres.

Lateral Column.—The degeneration of the C.P. tract is now well marked. It occupies a triangle, whose posterior base, separated from the posterior cornu by the thick layer of the lateral limiting zone, extends inwards from the periphery for about half the length of the posterior cornu. The apex reaches anteriorly to the level of the posterior commissure.

Anterior Column.—Normal.

Posterior Horn.—At the base of the posterior horn two or three shrunken cells can with difficulty be made out. There is a thin plexus of fine myelinised fibres anterior to the substantia spongiosa. The trabeculæ of the latter are thickened and numerous large Deiter's cells are visible. The vertical fibres, as well as the entering root fibres are degenerated.

Anterior Horn.—Apparently normal.

Clarke's Column.—This appears on each side as a faintly indicated white disc. Its posterior half is sclerosed. At its anterior pole, on each side, a thin plexus of fine fibres is visible, together with a few shrunken cells.

Lower Dorsal.—D. X.

Posterior Column.—There is still no trace of the differentiation of Goll's from Burdach's column. The whole breadth of the posterior column is occupied by the greyish white area of sclerosis. Some dark stained medullate fibres still occupy the apex and lateral boundaries of the column—in the site previously occupied by the cornu-commissural tract (ascending fibres from posterior roots?)—but these show an increasing number of degenerating fibres.

Lateral Column.—The patch of lateral sclerosis now takes on an extension forwards along the periphery of the column, the increase probably corresponding to the area of the direct cerebellar tract. This marginal degeneration reaches forward to a level corresponding to one-third of the peripheral circuit from posterior root to anterior fissure. The anterior portion of the degenerate tract is much less affected than the posterior.

Anterior Column.—There is now some disappearance of nerve tubes, with swelling and disintegration of others, and increase of interstitial tissue, in the anterior tips of Turck's columns on each side, where these border on the anterior fissure.

Posterior Horn.—The condition is the same as that described in the lumbar region.

Anterior Horn.—There is apparently some diminution in the number and volume of the internal group of motor cells.

Clarke's Column is completely sclerosed, both as to cells and fibres.

Mid-Dorsal. D. VII. to D. IV.

Posterior Column.—The narrow strip of medullated tissue bounding the commissure and posterior cornua shows a progressive loss of healthy fibres.

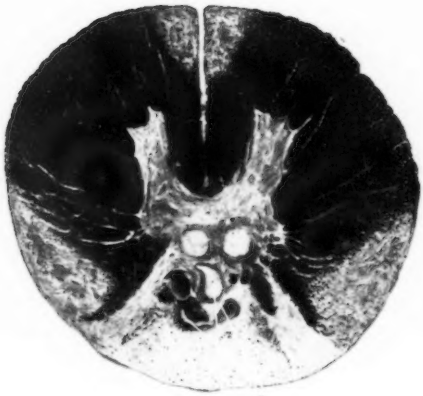
Lateral Column.—The degeneration in this column reaches its maximum extent about D. V. and at that level occupies a triangle, elongated anteriorly—whose base covers rather more than half the width of the lateral column, while the apex reaches the level of the summits of the anterior cornua. The lateral limiting layer is still preserved.

Anterior Column.—The sclerosis of the tips of Turck's column is well marked.

Posterior Horn.—This seems to be shrunken in size—the caput cornu reaching less than half way to the periphery. Its condition is as already described.

Anterior Horn.—No fresh changes are visible.

Clarke's Column.—Sclerosed.



a *b*

FIG. 3.

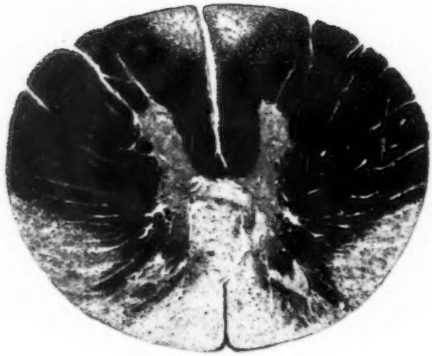


FIG. 4.

From D. VI. to D. III. an abnormality was present in the posterior column. A rounded patch of dense, sclerosed tissue occupied the anterior angle of the column, close to the commissure, displacing the fibres previously present there. These latter were represented in all probability in a ring-like whorl of medullated and partially degenerate fibres, which occupied the middle third of the column, posterior to the sclerosed patch already described. The whorl of fibres enclosed a second circular sclerosed area, and the remainder of the posterior column was occupied by sclerotic tissue as at former levels.

Upper Dorsal. D. II. to D. I.

The sclerosis of the *posterior column* here reaches its maximum, the remnant of stained fibres in the lateral tracts of Burdach's column, and posterior to the commissure, having undergone still further diminution.

Lateral Column.—The anterior marginal extremity of the sclerosed area does not extend so far forward as previously, having reached its maximum extent at the mid-dorsal region.

Anterior Column.—There is well-marked and symmetrical sclerosis of the anterior third of Turck's column. Degenerate tubes and increased neuroglia can be made out in the remainder of the direct pyramidal tracts on each side.

The shape of the cord departs from the normal here, being more oval than is usual at this level. This may possibly be due to antero-posterior shortening, caused by sclerotic changes in the anterior and posterior columns.

At the upper dorsal region, viz., D. I., there is apparent for the first time a distinct differentiation of Goll's from Burdach's column. A further change that takes place from this level onwards is the appearance of an increasing number of healthy, medullated fibres in the postero-external fields of Burdach's columns.

Lower Cervical. C. VIII. and VII.

Posterior Column.—The increase in the healthy fibres in Burdach's column now extends over the whole of that tract, and serves to distinguish it from Goll's tract. The increase in healthy fibres is also apparent in Lissauer's tract, which, though still much degenerated, yet contains more black-stained fibres than previously. Goll's column remains sclerosed up to the posterior commissure, and is shrunken to a narrow streak on each side the posterior fissure.

Lateral Column.—The position and degree of the sclerosis are as before. The anterior point reached by the degeneration in the D. C. T. is nearly on a level with the anterior commissure. In the upper sections of this region, however, there seems to be an increase in the thickness of the neuroglial trabeculæ in the position of Gower's tract, but without any distinct degeneration of the tubes.

Anterior Column.—There is well-marked and symmetrical sclerosis of Turck's column, appearing as a bluntly wedge-shaped tract on each side of the anterior fissure. The rest of the ground fibres are normal.

Posterior Horn.—This is distinctly atrophied and shrunken. There are a few nerve-cells in each cornu, but they appear below the normal size. The gleia cells are numerous and deeply stained. The preserved fibres with well-marked axis-cylinders, however, appear to be more numerous than previously.

Anterior Horn.—The inner or mesial group of cells has undergone diminution in number, and in size of the individual cells. The remaining groups, however, contain well-developed cells and processes.

Middle of Cervical Swelling. C. VI. to V.

An abnormality is again present in the posterior part of the cord at this level.

It commences at the level of C. VII. as a circumscribed oval-shaped area of sclerosed tissue, which occupies a position between the neck of the left posterior cornu and the adjoining portion of Burdach's column. The sclerotic area increases in size with successive sections, and reaches its maximum at the level of C. VI. In the position it occupies it displaces inwards towards the mesial line the streak of medullated fibres which has been described as bordering the internal margin of the posterior cornu. Its outer side is in contact with the lateral reticular formation. Completely sclerosed at its first appearance, at higher levels its area is traversed by scattered medullated fibres, a few of which are healthy but the greater number degenerated. By the level of C. IV. all traces of the abnormality have disappeared.

Posterior Column.—The condition is similar to that described previously. Burdach's column continues to receive an increasing number of healthy fibres, and also some degenerate ones. The healthy fibres are aggregated for the most part in the postero-external fields of the tract, adjacent to the posterior roots, and in Lissauer's tract. Goll's column also contains more myelinised

FIG. 5.

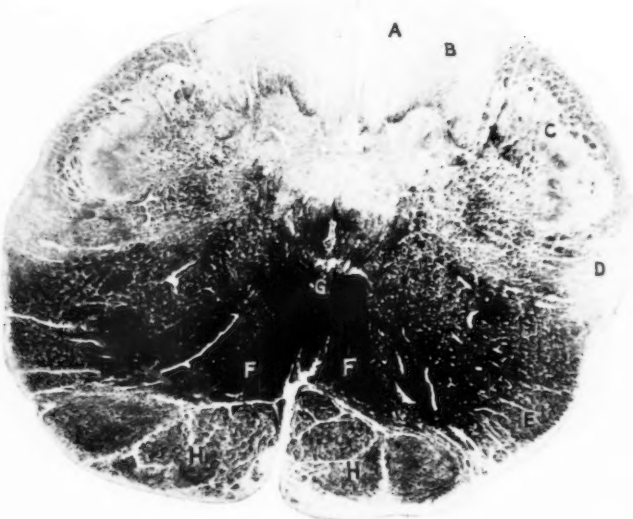


FIG. 6.

fibres than at former levels. These pass obliquely through the column towards the commissure, and many of them are degenerated.

Lateral Column.—The sclerosed area maintains its former position. The anterior extremity reaches further forward on the left side than on the right.

Anterior Column.—As before. The direct pyramidal tract on each side is symmetrically sclerosed. There is probably some increase of the neuroglia in the anterior ground fibres.

Posterior Cornua.—The abnormality on the left side has been already described. On the right side there is some improvement in the fulness of the fibrillar reticulum anterior to the substantia spongiosa.

Anterior Cornua.—There is asymmetry of the anterior horns, the right being larger than the left.

Upper Cervical. C. IV. to III.

Posterior Column. — Goll's tract is sclerosed throughout. Several fibres in advanced degeneration pass through it antero-posteriorly. These are more numerous near the commissure. Burdach's column, in its postero-external triangle, and Lissauer's tract contain a fair number of myelinised tubes, some of which contain healthy axis-cylinders.

Lateral Column.—The degenerate area is smaller in size than previously. The complete degeneration is limited to a small, oval area, which does not now reach to the periphery, and whose anterior limit does not pass beyond the level of the posterior commissure. The fibres of the D. C. tract, however, still contain many degenerated tubes.

Anterior Column.—Turck's columns still sclerosed.

Posterior Cornua.—Still degenerated and shrunken. The abnormality on the left side has now disappeared. Perhaps the fibrillar plexus is more stained than previously.

Anterior Cornua.—The right is still larger than the left. Both contain some shrunken cells in their anterior portions. The lateral groups of cells on each side are large and well developed.

Resumé of Cord-Changes.

Complete Sclerosis of Goll's Column from the sacral to the upper cervical region, most pronounced in the upper dorsal region.

Sclerosis of Burdach's Column (only recognisable as such above the level of the upper dorsal), the degeneration being constant in the portions adjoining Goll's column, and being less marked below in the parts bounding the commissure and the posterior cornua, and above in the postero-external fields.

Sclerosis of Lissauer's Tract less marked in the cervical region.

Sclerosis of the C. P. Tract, from lumbar region upwards, reaching its maximum at mid-dorsal level, and thereafter diminishing.

Sclerosis of D. P. Fibres, from mid-dorsal region upwards, increasing from below upwards.

Sclerosis of D. C. Tract, from lower dorsal region upwards. Most marked below.

Sclerosis of Posterior Horns and Posterior Roots throughout. Anterior horns well preserved, with the exception of a few cells of the mesial group and anterior group. Almost total escape of the anterior roots (to be described later).

Complete Sclerosis of Clarke's Column.

The condition of the nerve roots requires separate mention.

(A.) *Posterior Roots and Ganglia.*

These were examined: (*a*) above the ganglia; (*b*) in the ganglia; (*c*) below the ganglia; (*d*) in the mixed trunks.

The specimens were taken from the lumbar and dorsal region. I regret that the cervical roots were not preserved, all the more because the condition of the intra-medullary root-fibres in that region suggests that they would have shown, perhaps, a state of better preservation than those of the lower level. Cross-sections of the extra-medullary roots, however, were frequently included in sections of the cord in the cervical region, and an examination of these showed no marked difference between these roots in cross section and those belonging to other levels of the cord.

(*a*) The posterior roots between the ganglia and the cord showed extreme degeneration. Both by Marchi's and by Schäfer's method, only a very few dark-stained tubes of normal calibre were present in each root. In the unstained spaces between these larger fibres were seen fine small nerve-fibres, empty sheaths and connective tissue. The fine nerve fibres, probably the "embryonal" fibres of Auscher and Déjérine,[10] were frequently closely packed, in bundles of from six to twenty fibres, the individual

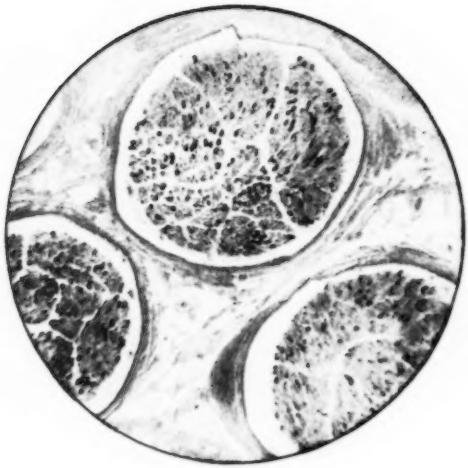


FIG. 7.

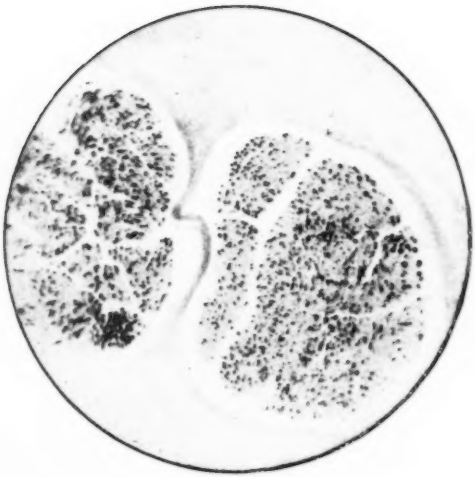


FIG. 8.

tubes measuring from 3 to 8 in diameter. Extremely fine, but well-stained axis-cylinders were present in most of them, and generally a very thin myelin sheath, but the latter remained unaffected by myelin stains such as Schäfer's and Pal-Weigert's. In others, the myelin seemed disintegrated and granular, and the axis-cylinders were sometimes hypertrophied, sometimes absent. The empty nerve-sheaths, both of the large and of the fine fibres, were recognised as rings, either round or distorted by pressure, their outlines being frequently marked in Marchi and osmium preparations by a marginal ring of fine black granules.

(*b*) In the spinal ganglia both ganglion cells and nerve fibres call for notice.

The cells, as indeed the ganglia themselves, appeared smaller than normal. Measurements through their longest diameters (when not perfectly round) gave their average diameters as 50μ, the limits between which they varied being from 25μ to 70μ. This is below the figure given by Lenhossék[38] for the average diameter of healthy cells of the larger sort, viz., 60 to 80μ. Striking was the amount of pigmentation they displayed. In very few was this limited to a mass lying between the nucleus and the thickened peripheral layer of cell plasma, or to a crescentic ring investing the nucleus. On the contrary, in the majority of the cells it occupied approximately two-thirds of the area of the cell plasma, obscuring the nucleus completely. It consisted of coarse pigment granules, insusceptible to stain, and agglomerated in many cases into dense clumps in which no differentiation of separate granules could be made out.

The cell plasma was invariably opaque and turbid, never in the least degree brightly refractile or translucent. It stained readily with plasma stain and then showed itself finely granular in structure. The finer details of the granules could not be made out owing to shortcomings in the staining, due to my want of familiarity with Mann's method. No vacuolation was present, but the normal marginal space between cell plasma and capsule figured and described by Lenhossék was frequently present. The nuclei were occasionally well defined as also were the nucleoli, but not in every cell. The cell processes also could only seldom be distinguished. The epithelium of the capsules appeared normal.

I am unable to venture on an opinion as to how far these appearances are abnormal or the expression of artefacts. The latter explanation readily suggests itself in connection with the shrinking in size. But, on the other hand, the method of harden-

ing used by Lenhossék, from whom the normal measurements are taken, viz., corrosive sublimate and alcohol, would cause as much shrinking as the Müller's fluid and alcohol used in my own preparation. The amount of pigmentation that may be considered normal is another vexed question. Wollenberg, who discusses the matter,[22] apparently concludes, with Obersteiner, that the decision as to what is and what is not pathological is frequently an arbitrary one. The opacity of the cell plasma may —according to Trzebinski (quoted by Wollenberg, *loc. cit.*)—be produced by the Müller's fluid and alcohol employed. I can speak definitely only as regards vacuolation and fatty degeneration of the plasma, neither of which could be recognised in these sections.

On the whole, a comparison with the few normal specimens I possess leads me to the opinion that the ganglion cells were smaller and more pigmented than average normal cells. Whether this, however, corresponds with the pigmentary atrophy described by other observers, I am unable to say.

In the fasciculi of transversely divided nerve-fibres which appeared in the sections of the ganglia, it was easy to recognise marked degeneration. Numerous lacunæ were present, corresponding to spaces vacated by swollen medullary sheaths that had disappeared in handling. Many of the fibres showed granular and disintegrated myelin, and from many the axis cylinders had disappeared. Between these were spaces occupied by connective tissue, fine fibres, some apparently healthy, some undergoing degeneration, and empty Schwann's sheaths.

Logwood preparations showed a distinct multiplication of connective tissue nuclei, both in the endoneurium between the nerve fibres and in the interstitial tissue between the ganglion cells.

(*c*) *Below the Ganglia.*—The roots showed the same condition of degeneration as already described.

(*d*) *The Mixed Trunks.*—Tranverse sections here showed the two roots lying within a connective tissue-sheath apparently much too large for its contents.

The constituent portions of the nerve were easily recognised, in Pal-Weigert and Schäfer preparations, the anterior root showing, for the most part, regular and well stained fibres of uniform size, while the posterior showed a few dark stained discs and rings in the midst of a mass of unstained sclerotic tissue.

The Anterior Roots.—These consisted, for the most part, of large nerve tubes, of 10-12μ in diameter, with healthy sheaths and axis cylinders. They were not wholly free, however, from degenerative change. This affected chiefly the myelin sheath.

In some the myelin had separated from the Schwann's membrane and appeared loose in the interior of the tube. In others it was aggregated into a mass lying against the inner wall of the sheath. In others, again, the myelin appeared to have become absorbed, leaving a naked axis cylinder inside the tube so formed. This condition, well shown in benzo-purpurin and Mallory-hæmatoxylin preparations, appeared to resemble the denudation of the axis cylinder described by Babinsky and others as occurring in multiple sclerosis. A few fine fibres in good preservation were present in the anterior roots, but in much smaller numbers than in the posterior.

The Peripheral Nerves.

Those examined were the sciatic in the upper part of the thigh, the anterior crural, anterior tibial, internal plantar, median, ulnar, radial and posterior interosseous. Each and all of these showed extreme degeneration. By Schäfer's method not only the healthy medullated sheaths, but those also which contained degenerate but unabsorbed myelin, gave the black reaction. The unstained areas, therefore, corresponded to parts which were totally devoid of myelin, either healthy or degenerate.

To obtain a comparative estimate of the degrees of this degeneration various methods were adopted.

A modification of Sakaky's method[26] was used for estimating the number of healthy fibres in the fasciculi. By means of a mm. stage micrometer and a corresponding ruled ocular the number of healthy fibres present in a series of squares each with a side measurement of ·1 mm. was counted, and an average was taken from nine or sixteen of these squares. Observations on healthy nerve fibres gave 32 nerve fibres of 10 to 12μ each in diameter as the average normal contents of such a square. With the normal figure of 32 was compared the number of healthy fibres per ·1 mm. square in the best preserved portion of the cross section of the nerve. The small fibres, of which large numbers were apparent in all of the peripheral nerves, were excluded from the calculation on account of the difficulty of getting any satisfactory estimate of their number in normal nerves.

As regards the larger fibres, it was found that in the majority of the nerves examined, viz., in the median, radial, posterior interosseous, sciatic, anterior crural, and internal plantar, occasional fields could still be found in which a few squares reached or approximated to the normal content of fibres.

In the ulnar and anterior tibial, however, in not a single field

in the cross section of the nerve could an area containing 32 healthy fibres per square ·1 mm. be found.

The relative degree of sclerosis was roughly estimated by measuring the largest superficial areas in each nerve in which not a single healthy medullated fibre could be found.

The results may be thus tabulated:—

In the posterior interosseous nerve a whole field measuring ·3 mm. in diameter	contained not a single healthy fibre.
In the ulnar, a space measuring ·3 × ·15 mm.	" " " " "
In the radial, a space measuring ·2 × ·2 mm.	" " " " "
In the median, a space measuring ·18 × ·18 mm.	" " " " "
In the anterior tibial, a space measuring ·175 × ·175 mm.	" " " " "
In the internal plantar, a space measuring ·125 × ·15 mm.	" " " " "

In the sciatic and anterior crural nerves no sclerotic area larger than ·1 mm. square void of healthy fibres could be found.

Contrary to one's expectation, it thus appeared that the greatest destruction of nerve fibres had taken place in the mixed nerves of the upper limb. In the nerves of the lower limb it appeared that portions more peripherally placed suffered greater degeneration than those nearer the cord. Connective tissue hyperplasia was extreme in those fibres in which the sclerosis was most marked, and reached its maximum in the internal plantar. In the same nerve the accompanying artery showed an extreme thickening of the intima, the lumen of the vessel being almost obliterated thereby. In the sciatic and anterior crural there was a great deposit of fat in the interfasicular connective tissue.

The degeneration in every case was of the ordinary parenchymatous type, the medullary sheaths being either granular, broken into clumps, disintegrated, with formation of droplets, or absorbed. Persistence of the axis-cylinders, such as was observed in the anterior roots, was nowhere seen in the peripheral nerves.

The Medulla.

Sections were cut from the whole length of the medulla with the exception of the region of the pyramidal crossing, which was unfortunately destroyed in removing the parts. The changes met with may be sufficiently described at three levels.

(*a*) Between the pyramidal decussation and the lower level of the olives.

The longitudinal fibres at this level comprise the funiculi graciles and cuneati, the lateral and pyramidal columns, and the two narrow layers which, lying on each side of the middle line dorsal to the anterior pyramids, represent the fibres of the inter-olivary layer or fillet.

It is these latter fibres and the adjoining tracts of the antero-lateral ground fibres which, alone of the longitudinal bundles, present healthy fields of well-stained fibres at this level. Of the remainder, the funiculus gracilis (Goll's column) maintains its former condition of almost complete sclerosis. The funiculus cuneatus (Burdach's column), better preserved than the preceding, shows a slender band of stained fibres along the margin which adjoins the central grey matter. In the lateral columns there is a general lightening of the stain, amounting in the region of the D. C. tract to complete sclerosis. Gower's tract shows here more definite signs of degeneration than were apparent in the cord. In the pyramids, there is a general faintness of the medullary stain, and, as was to be expected from the condition of the direct and crossed pyramidal tracts, numerous degenerated fibres are present.

Of the grey matter, the clavate nucleus is practically absent, being represented only by a very few rounded nerve cells.

The cuneate nucleus is also poorly developed, being considerably below its normal bulk. The caput cornu posterioris, however, now swelling into the tubercle of Rolando, appears to be recovering from its former atrophy, and shows a well-developed head of grey matter and numerous small, nucleated cells. The central canal appears normal. The ependymal lining is distinctly columnar, and outside the epithelial layer is a loosely-arranged gleial network, with numerous Deiter's cells.

The healthy and well-developed condition of the fillet layers already referred to occasioned some surprise, on account of the meagre development of the innermost strands of the internal arcuate fibres. These latter, proceeding from the nuclei of the posterior columns, bend round the central grey substance, and after decussating give rise to the fibres of the fillet layers. Owing probably to the sclerosis present in the clavate nucleus and the poor development of the cuneate nucleus, the strands passing from these nuclei to the fillet decussation are less full and less numerous than in a normal medulla. Some bundles of well-stained arcuate fibres which are present arise from the neighbourhood of the posterior horn, and occupy the outermost division of the internal arcuate fibres.

(*b*) Lower level of the inferior olive.

The healthy fibres of the fillet layers now appear as the well-stained interolivary layer, and, dorsally to these, the median area of the reticular formation is occupied by well-stained fibres, which, according to Ferrier,[43] are contributed from the anterior root-zone of the cord. The lateral area of the reticular formation is less well preserved. Towards the dorsal margin of the lateral column there is a considerable accession of healthy, dark-stained fibres apparent in cross section, and these are evidently related to the dark-stained bundles of internal arcuate fibres which are seen to proceed to the region of the corpora restiformia from the olives and the fillet.

The clavate nucleus is still absent as such from the postero-internal column, but traces of grey matter with infrequent round cells can be made out in its site.

The caudate nucleus is now well developed, and contains numerous nerve cells.

The olivary bodies are well developed, and the numerous fibres passing from them show the black stain of normal myelin.

The central grey matter and the posterior longitudinal fasiculus also appear normal.

The pyramids, however, are smaller than normal, and still contain many degenerated fibres.

(*c*) Upper level of olive (at and above calamus scriptorius).

The prominent feature from this level upwards is the gradual increase in the healthy fibres of the corpus restiforme of each side by the continued accession of arcuate fibres from the olivary bodies.

There still appears a small area of sclerosis in the site occupied by the direct cerebellar fibres. The pyramids continue of smaller bulk than normal, but they exhibit less evident signs of degeneration than at lower levels.

The cranial nuclei (XII. and X.) appear to be normal. The fibres of the fillet and of the posterior longitudinal fasiculus are well stained and healthy.

From this level upwards the medulla and pons exhibit no marked morbid change.

Cerebellum.

No signs of degeneration or maldevelopment could be found in the cerebellum. The inferior peduncle showed healthy fibres and axis-cylinders throughout. The plicæ showed a normal

arrangement, with a well-marked nuclear layer, and numerous healthy Purkinje's cells.

Remarks on the Case.

In its clinical features and its post-mortem appearances, the case reported shows no peculiarity. With the former I do not intend to deal, as the matter has been so recently and exhaustively treated by Mackie Whyte in this Journal.[42]

None of the pathological conditions described are new to the literature of the disease, with the trivial exception, perhaps, of the abnormal arrangements described as occurring in the posterior column of the cord, in the upper dorsal and mid-cervical regions.

In many features the case bears a close resemblance to some of the more recently reported cases, *e.g.*, those of Simon (16, in summary of autopsies), Bonnus (*ib.* 17), Mirto (*ib.* 13), and Guizetti (*ib.* 12). In four of these, viz., 12, 16, 17, and my own, the degeneration of the posterior column reached its maximum in the dorsal region. In the same four cases the cornu-commissural tract, and fibres representing Flechsig's oval tract, escaped injury either relatively or entirely.

The degeneration of the crossed pyramidal and direct cerebellar tracts, recorded in nearly every reported case, was present in these cases too. In addition Lissauer's tract was more or less affected in each, as it was also in cases 11 and 10.

In all, as indeed in every recorded case where they have been examined, the posterior roots were sclerosed. The condition of the spinal ganglia is not mentioned in cases 16 and 17. In 12 and 13, however, as also in Blocq and Marinesco's case, 11, and probably also in my own, 18, changes were present. Interstitial hyperplasia, with changes in the ganglion cells, vacuolation, or shrinking, are mentioned in every case.

In the medulla, sclerosis of the posterior columns, with degeneration of nuclei, is recorded in cases 12, 16, 17, 18, and also in 11, and degeneration, in the bulb, of the direct cerebellar fibres, is also recorded in cases 11, 12, 16 and 18.

Similar changes in the posterior columns of the medulla are reported by Friedreich (cases 2 and 4), and were also observed in the doubtful cases of Brousse and Tedeschi.

Some degeneration of the fibres of the anterior pyramids occurred in Guizetti's case and my own.

The cerebellum has been found normal in every case in which its condition has been recorded, with the exception of Auscher's doubtful case.

The peripheral nerves were atrophied or degenerated in cases 12, 13, 17 and 18, severely so in the last case.

In the light of these cases, together with the fact that similar changes have been recorded by Friedreich and by Rutimeyer, it must be admitted that degeneration of the peripheral nerves, which may reach an extreme degree, ought in future to find mention in any complete account of the morbid anatomy of Friedreich's disease.

The nature of the fine fibres observed in the peripheral nerves in this as in other cases of Friedreich's disease is a question of great interest.

Auscher[10] describes those present in his case as fibres having embryonal characters, *i.e.*, with an axis-cylinder, and either a very slight myelin sheath or none at all. He expressly states that he found no degenerated tubes nor empty sheaths.

Guizetti[11] records the presence of numerous fine fibres with a diminution in the number of coarse fibres. He describes and figures various histological conditions of the fine fibres in the nerves and the posterior roots, viz., some—a majority—with axis cylinders, some without, some apparently deprived of medullary sheaths, and others in which the sheaths stained slightly with osmic acid. He assumes these conditions to be the result of atrophy.

In my own case several varieties of fine fibres could be distinguished, viz. :—

(*a*) Fine fibres with a slender axis-cylinder and an apparently medullated sheath. I say *apparently* because the substance of the sheath, though colouring with plasma stains, and very faintly also with chromatin stains, yet

failed to take up the specific myelin stains, such as those of Weigert and Schäfer. This fact has been noted also by Tedeschi.[15]

(*b*) Fine fibres showing more or less traces of degeneration, *e.g.*, (*a*) a granular condition of the sheath, (*β*) hypertrophy of the axis cylinder, either with or without a thin investment of myelin, or (*γ*) disappearance of the axis cylinder.

(*c*) Empty fibres, myelin and axis-cylinder having undergone absorption, the Schwann's sheaths remaining having frequently fine granules, staining black with osmium, attached to their inner surfaces.

All these appearances have been noted and described in the degeneration of peripheral nerves which accompanies various scleroses of the cord. Westphal figures them in a case of postero-lateral sclerosis (*Archiv. für Psychiat.*, Bd. viii., taf. xi.), and again in a case of central and peripheral degeneration due, he thinks, to tubercular infection (*ib.*, Bd. xvi., taf. xi.). Sakaky,[26] and Oppenheim and Siemerling,[28] describe them in the peripheral neuritis accompanying cases of tabes. Many observers have described the arrangements of the fine fibres as being in closely packed clusters or fasciculi, as if held together by some outer investment. Oppenheim and Siemerling (*loc. cit.*) describe bundles of well-preserved rods of fine calibre inside the empty Schwann's sheaths of larger fibres. These observations suggest the presence of fibrils of regeneration rather than that of atrophied fibres.

Since Déjérine has shown[29] that the peripheral degeneration in some cases of tabes attacks first the distal extremity of the peripheral neuron, and may co-exist with complete integrity of the spinal ganglion and posterior spinal root, it is reasonable to suppose that the peripheral degeneration in cases of sclerosis of the cord, such as those alluded to above, may be akin to that which occurs in the toxæmic states due to lead, alcohol, diphtheria, &c. In these, as Gombault,[37] S. Mayer,[38] Pitres and Vaillard,[39] Gudden,[24] and others have described, there occurs, after the degenerative period, an outbudding of small regenerative fibres ("seg-

ments intercalaires," "schaltstucke"), which, in cross-section, would present the characters of the fine fibres described under (*a*). Indeed, it seems difficult to account in other ways for the presence of these fine medullated and cylindered fibres in the midst of otherwise general degeneration. If Fleming's view[31] be correct, that the normal fine fibres in peripheral nerves are the first to undergo degenerative changes, the fine fibres under discussion can hardly represent the original fine fibres present normally in every peripheral nerve, and unless simple atrophy of nerve fibres can take place without a trace of molecular change or disintegration, it is equally difficult to conceive these fine fibres as originating in the atrophy of the larger ones. It seems, at least, equally probable that they may be single or multiple regenerative fibres, the outcome of the segmental neuritis of larger tubes. Further, there is no cause hindering the degeneration in their turn of the fine regenerative fibres. As Gudden remarks[24] (p. 731):—"One factor only need be assumed, viz., that the patient is a debilitated individual, and that the toxic virus does not at once escape from the system. In such a case the restitution, in the first place, of the nerve fibres, proceeds slowly, and secondly, these latter, when scarcely in working order, either through the feebleness of the organism or through the presence of some fresh virus. . . . are checked in their further development and again fall into decay." Gudden instances as an illustration of "fresh virus" the infection of tubercle, and it is noteworthy that if the doubtful cases of Friedreich's disease in the appended list be included, a history of tubercle is present in seven out of the twenty-two autopsies recorded.

Such a process as is suggested above would well account for the appearances found in the fine fibres in classes (*b*) and (*c*). It may be objected to this explanation of the presence of the fine fibres that the primary pathological processes, central and peripheral, in Friedreich's disease, have not been shown to depend on any toxæmic dyscrasia, inherited or acquired. The same objection, however, applies equally to amyotrophic lateral sclerosis, and to the

hereditary form of muscular atrophy. These diseases affect the peripheral motor neurons, much, it appears to me, as Friedreich's disease affects the peripheral sensory neurons associated with muscle. In both these forms of muscular atrophy Gombault has observed [37] in the degenerate peripheral nerves the occurrence of segmental neuritis with regenerative processes.

The extreme preponderance of the degeneration in the posterior roots rather than the anterior indicates that it is the afferent fibres of the peripheral nerves that are chiefly attacked. The fact, moreover, frequently recorded, that the patients' sensibility to touch, pain, and temperature remains unimpaired throughout, suggests that it is the afferent nerves from muscles that chiefly suffer. This agrees with Guizetti's conclusion, who was of opinion that in his own case there existed a systematic atrophy of sensory fibres. In his case, however, he considered that muscular and cutaneous fibres were both affected, and it should be remembered that, clinically, the patient's tactile and thermic sensibility were diminished below the knees, and that some tactile impairment was present in the hands.

The presence of abnormal arrangements in the structure of the cord which I have described as occurring in the dorsal and cervical portions is suggestive of structural anomaly dating back to the developmental period of the cord. Sections containing these abnormalities were variously stained in the endeavour to ascertain the finer structure of the growths. The nerve fibres traversing the areas were invariably degenerated, and none of them possessed axis cylinders. The intervening ground substance resembled closely the sclerotic tissue present in the posterior parts of Goll's column, and its density opposed a serious obstacle to its examination. The felt-like tissue composing it shrank extremely in the process of dehydrating and clearing, so that thin sections were destroyed by the tearing consequent on the shrinkage. Deiter's cells, however, could not be distinctly recognised, but corpora amylacea were present in considerable numbers. The staining reactions were those of glial tissue, and in all probability the structure was that of

a circumscribed gliomatous mass of long standing, in which the cellular elements seemed to have disappeared.

Finally, no appearance at all corresponding to Déjérine's description of whorls of glial tissue could be recognised in this case. The grey sclerotic areas in the posterior and lateral columns were found histologically to have the usual structure of neuroglial hyperplasia elsewhere. Numerous large Deiter's cells were present, frequently of elongated or lanceolate shape, as if obliquely-lying elongated cells had been divided in a plane approximating that of their longest diameter. In all, or nearly all, the faintly stained granular nucleus could be recognised, and in some the processes could be traced for a short distance from the cell. These elongated cells and smaller rounded ones formed dark-stained masses in a dense spongy-looking groundwork, the minute dark points in which probably corresponded to gleial fibres in cross-section. The cells were least distinct, and stained less freely in the older and more completely sclerosed areas, while they stained most distinctly in the transition zones between healthy and degenerating tissue. Where they stained least distinctly, *e.g.*, in the peripheral portion of the posterior columns, there numerous corpora amylacea were thickly strewn, and the converse of this arrangement held equally true, viz., the corpora amylacea were least numerous in those regions where the glial cells were most distinct. The same histological features characterised the areas of degeneration in the various columns of the cord. No special peculiarities could be made out in the lateral areas of sclerosis. The vessels and capillaries were nowhere markedly thickened, but were everywhere distended with blood cells.

The last mentioned fact is of interest in relation to the vascular theory of the causation of the disease which was favoured by Pitt, and by Blocq and Marinesco. These observers found changes in the blood vessels of the posterior columns, as also did Schultze in his second case, and Everett Smith.

On the other hand such changes were absent in the cases of Rutimeyer, Déjérine and Letulle, Guizetti, and in my own.

Guizetti, who discusses this matter, points out that disease of the posterior vascular system of the cord would leave unexplained the condition of the posterior roots and spinal ganglia, and that Clarke's column, which is invariably sclerosed, is supplied by the anterior system of vessels. Moreover, that such vascular dilatations as Pitt and Blocq and Marinesco have recorded, are a frequent accompaniment of sclerosis of the cord, and probably secondary to it.

On the whole it appears that our present knowledge of the pathogenesis of the disease cannot be better summed up than in Guizetti's conclusion, that Friedreich's disease depends upon a congenital predisposition, in consequence of which, in the early years of life, certain systems of fibres and nerve cells undergo a process of progressive atrophy, and that the process is independent of any contributory effect from vascular alterations.

To Guizetti's conclusion might be added that by "certain systems of fibres and cells" ought perhaps to be understood the following :—

(*A*) (Due to arrested development at the eighth month?) Tracts in the cord which are the latest to undergo medullation, viz., the pyramidal tracts, direct and crossed, and the postero-internal tract.

(*B*) (Secondary to mal-development of Goll's column?) Systems of fibres and cells functionally related to the postero-internal tract. The extent of the atrophic process as to systems attacked, and degree of degeneration in individual systems, may be dependent upon the duration of the disease. These systems may include :—

(*a*) The peripheral sensory neuron complex in its entirety, viz., peripheral sensory fibres, ganglion cells, posterior roots and root-zones (Burdach's, Lissauer's), fibres to Clarke's columns, to the middle zone, and to the anterior cornua.

(*b*) Portions of the central sensory neurons, viz., cells in posterior horns, with associated ascending fibres, cells in Clarke's columns, with associated fibres (direct cerebellar tract), cells in middle zone, with associated fibres (Gower's tract), cells in medullary nuclei, clavate and cuneate, with associated fibres (internal arcuate).

The atrophic changes in every system are arrested in the medulla, when the level of the lower olive is reached.

I append a summary in tabular form of the pathological conditions present in the autopsies of Friedreich's disease to which I have been able to refer, and I believe the list is fairly complete up to the date of its compilation (July, 1898). A useful summary of most of the cases included was given by Tedeschi [15] in 1896. I have excluded from the list all cases of the cerebellar type of the disease with which Nonne, Menzel, Marie, Sanger Brown and others have made us familiar, because it is clear—*pace* Senator—that these form a group quite distinct, clinically as well as pathologically, from the type described by Friedreich.

I have also placed in a separate list the doubtful cases of Kahler and Pick, Brousse, Auscher, and Tedeschi.

Kahler and Pick's case presents some close resemblances to hereditary ataxia. The age at onset—on menstruation, at 16—the general course of symptoms, the loss of knee-jerk, the impaired speech, the preserved pupil reflex, and the ataxia, all agree with Friedreich's symptom-complex. In addition spinal curvature was present, and a sister of the patient is said to have been affected similarly to herself.

On the other hand, Westphal (*Archiv. für Psychiat.*, Bd. ix., p. 697), draws attention to the early onset of paralytic weakness in the case, with outstretched legs and pointed toes, as dissociating it from Friedreich's group. In addition, nystagmus was absent, and there is no mention of choreiform movements, or of pes cavus.

Very similar to Kahler and Pick's case is that of Erlicki and Rybalkin [40] affecting also a young girl soon after puberty. In this case the rude strength of the legs was well maintained during the early period, and the majority of the symptoms of Friedreich's disease were present. On the other hand, deformity of the feet and spine were absent, as also were nystagmus and speech affection. More important, perhaps, was the hurried course of the disease, the symptoms developing with undue rapidity, and death ensuing from tubercular diarrhœa in twenty months from the onset. For this reason I have omitted the case.

Brousse's and Auscher's cases both concern women of later age, 24 years and 24½ respectively. In the former neither spinal nor foot deformity was present, nor, apparently, nystagmus. In the latter the reflex to light was absent, and post-mortem, brain and cerebellum were found of small size, while the lateral columns of the cord were unaffected.

In Tedeschi's case, as Mackie Whyte has pointed out,[42] many important clinical details are unfortunately omitted, *e.g.*, the condition of the knee-jerks, state of the spine, &c. The onset of the symptoms was anomalous, the arms being attacked before the legs, the gait was tabetic in character rather than cerebellar, and the patient was insane.

A case reported by Mitchell Clarke[41] has also been omitted because the complication of cerebellar tumour which was present vitiates the pathological record for purposes of comparison.

It only remains for me to express my grateful acknowledgments to Sir T. Grainger Stewart, who suggested to me the examination of the peripheral nerves, and to Dr. Eurich of Bradford, for kind assistance in the technic of neuroglia staining.

SUMMARY OF AUTOPSIES ON UNDOUBTED CASES.

Case 1.

A.—*Name and Duration.*—Andreas Lotsch; 15 years ill. Died at 38; cause, typhoid.

B.—*Author and Reference.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii., s. 1, 1863.

C.—*Cord.*—Small.

D.—*Membranes.*—Dural sac contained fluid. Pia milky and thickened, soft adhesions to cord.

E.—*Posterior Column.*—Grey, translucent, firm, from above lumbar swelling to calamus. Sclerosis most marked above lumbar swelling. *Microscopically*—Atrophy of nerve tubes and presence in their place of a finely fibrillar connective tissue—made up, in part, of the shrunken Schwann's sheaths. Between these elements a finely granular ground substance, clearing with acetic acid, and then revealing many round and oval nuclei.

F.—*Lateral Column.*—Normal.

G.—*Anterior Column.*—Normal.

H.—*Posterior Horn.*—Normal.

J.—*Anterior Horn.*—Normal.

L.—*Sp. Roots.*—Posterior roots atrophied.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*
O.—*C. Canal.*—A double canal containing fluid in cervical region.
P.—*Med.*
Q.—*Cbl.*
R.—*Cbr.*
S.—*Remarks.*

Case 4.

A.—*Duration.*—Fr. Suss; duration 27 years. Died at 42 of typhoid.
B.—*Author.*—Friedreich and Schultze. *Virchow's Archives*, Bd. lxx., s. 140.
C.—*Cord.*—As in former cases.
D.—*Memb.*—Pia thickened and opaque, and adherent to dura.
E.—*P. Col.*—Sclerosed throughout. In cervical region Goll's column chiefly attacked.
F.—*L. Col.*—Sclerosed, in posterior part, throughout whole length of cord. A small band of healthy tissue intervening between sclerosed tissue and posterior column.
G.—*A. Col.*—In the cervical region severe sclerosis of the right pyramidal tract, adjoining the anterior fissure. This extended upwards beyond the decussation, but did not extend downwards into dorsal region.
H.—*P. Horn.*—Atrophied.
J.—*A. Horn.*—Atrophy of grey substance at cervical swelling. Ganglion cells fewer and smaller than normal.
K.—*Clarke's Col.*—Cells atrophied.
L.—*Sp. Roots.*—Posterior roots atrophied.
M.—*Sp. Ganglia.*—Normal.
N.—*Peri. Nerves.*—Normal.
O.—*C. Canal.*
P.—*Med.*—Small. Sclerosed in Goll's and Burdach's columns, with atrophy of the ganglion cells of both nuclei. Anterior pyramids normal. Connective tissue hyperplasia, with corpora amylacea, in corpora restiformia as high as the level of cal. script.
Q.—*Cbl.*—Normal.
R.—*Cbr.*—Normal.
S.—*Remarks.*—Walls of vessels thickened, especially in neighbourhood of posterior column.

Case 5.

A.—*Duration.*—Charlotte Lotsch; duration 35 years.
B.—*Author.*—Schultze. *Virchow's Archives*, Bd. lxxix., s. 132.
C.—*Cord.*—Thin and flattened.
D.—*Memb.*—Pia thickened and opaque.
E.—*P. Col.*—Sclerosed. In the cervical region a strip of less degenerate substance, 1 mm. broad, remains, dorsal to the posterior commissure. Postero-external fields also contain some normal fibres.
F.—*L. Col.*—Posterior parts sclerosed up to the periphery, but the degeneration did not extend so far forward as in case 4.
G.—*A. Col.*—Both sides sclerosed for some distance from decussation downwards, in a comma-shaped tract.

K.—*Clarke's Column.*—Normal.
L.—*Spinal Roots.*—Posterior roots atrophied.
M.—*Spinal Ganglia.*—No mention.
N.—*Peripheral Nerves.*—Some atrophy in sciatic. Less in nerves of cervical and brachial plexus. More intense degeneration in hypoglossi.
O.—*Central Canal.*—No mention.
P.—*Medulla.*—Corpora restiformia slightly attacked.
Q.—*Cerebellum.*—Normal.
R.—*Cerebrum.*—Normal.
S.—*Remarks.*—Walls of capillaries in affected areas show fatty globules.

Case 2.

A.—*Duration.*—Justinie Suss; 15 years ill. Died of typhoid at 31.
B.—*Author.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii. s. 1, 1863.
C.—*Cord.*—Small.
D.—*Memb.*—Pia thickened and adherent to dura and to cord.
E.—*P. Col.*—Sclerosed between lumbar and cervical swellings. Above the cervical swelling only a thin zone, next periphery affected. Postero-median fissure obliterated.
F.—*L. Col.*—Sclerosed between lumbar and cervical swellings in the parts adjacent to the posterior column, but to a less extent than the posterior column.
G.—*A. Col.*—Normal.
H.—*P. Horn.*
J.—*A. Horn.*
K.—*Clarke's Col.*
} Grey matter of cord sclerosed in dorsal region.
L.—*Sp. Roots.*—Posterior roots atrophied.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*—Interstitial hyperplasia in the nerve trunks of the extremities.
O.—*C. Canal.*—Occluded with small cells.
P.—*Med.*—Sclerosed in posterior column.
Q.—*Cbl.*—Normal.
R.—*Cbr.*—Normal.
S.—*Remarks.*

Case 3.

A.—*Duration.*—Salome Suss; duration 14 years. Died at 28 of typhoid.
B.—*Author.*—Friedreich. *Virchow's Archives*, Bd. xxvi., s. 391 and xxvii., s. 1, 1863.
C.—*Cord.*—As in former cases.
D.—*Memb.*—Pia thickened and opaque, and adherent to dura.
E.—*P. Col.*—Sclerosed throughout, most marked in dorsal region.
F.—*L. Col.*—Sclerosed, in dorsal region, in posterior part of lateral column on left side.
G.—*A. Col.*
H.—*P. Horn.*
J.—*A. Horn.*
K.—*Clarke's Col.*

J.—*A. Horn.*—Unaffected.
K.—*Clarke's Col.*—Sclerosed in some sections.
L.—*Sp. Roots.*—Posterior sclerosed; anterior normal.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*—Radial and anterior crural showed no degeneration.
O.—*C. Canal.*—Double canal present at decussation of pyramids.
P.—*Med.*—Marked degeneration of funiculus gracilis; less severe of funiculus cuneatus. Some degeneration in funiculus rotundus.
Q.—*Cbl.*
R.—*Cbr.*
S.—*Remarks.*—Pitt associates the disease with a tendency to vascular degeneration and an ill-developed spinal cord.

Case 8.

A.—*Duration.*—H. Kern; duration 14 years. Died at 20.
B.—*Author.*—Rutimeyer. *Virchow's Archives*, Bd. cx., s. 215.
C.—*Cord.*—Small.
D.—*Memb.*—Pia thickened, numerous adhesions, and opaque.
E.—*P. Col.*—Sclerosed. Severe of Goll's column; less so of Burdach's. Escape of Lissauer's tract and of Flechsig's oval tract.
F.—*L. Col.*—Sclerosis of crossed pyramidal and direct cerebellar tracts, but less severe than that of posterior column. Escape of lateral limiting layer.
G.—*A. Col.*
H.—*P. Horn.*
J.—*A. Horn.* } Grey matter of cornua appears normal.
K.—*Clarke's Col.*—Sclerosed.
L.—*Sp. Roots.*—Posterior sclerosed, most markedly in dorsal region.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*—Marked degeneration of sciatic and median.
O.—*C. Canal.*
P.—*Med.*—Small. Sclerosis of funiculi graciles and cuneati.
Q.—*Cbl.*
R.—*Cbr.*—Normal.
S.—*Remarks.*

Case 9.

A.—*Duration.*—B. Kern; duration 9 years.
B.—*Author.*—Rutimeyer. *Virchow's Archives*, Bd. cx., s. 215.
C.—*Cord.*—Small.
D.—*Memb.*—No sub-dural exudation; pia thickened.
E.—*P. Col.*—Sclerosed; with exception, in cervical and lumbar regions, of small fields, as in former case.
F.—*L. Col.*—Sclerosis of crossed pyramidal and direct cerebellar tracts, with escape of lateral limiting layer. Some marginal degeneration extends forward beyond region of D. C. T. (Gower's tract?) Lateral sclerosis most marked in the cervical region.
G.—*A. Col.*
H.—*P. Horn.*
J.—*A. Horn.*
K.—*Clarke's Col.*—Sclerosed.

H.—*P. Horn.*—No mention.
J.—*A. Horn.*—No mention.
K.—*Clarke's Col.*—Sclerosed.
L.—*Sp. Roots.*—Posterior sclerosed.
M.—*Sp. Ganglia.*—No abnormality.
N.—*Peri. Nerves.*
O.—*C. Canal.*
P.—*Med.*—Normal, except for some hypertrophy of connective tissue.
Q.—*Cbl.*
R.—*Cbr.*
S.—*Remarks.*

Case 6.

A.—*Duration.*—Clara S.
B.—*Author.*—Everett Smith. *Boston Medical and Surgical Journal*, October, 1885.
C.—*Cord.*—Asymmetrical, small.
D.—*Memb.*—Meninges injected and adherent to bone.
E.—*P. Col.*—Sclerosed; with exception of a small area lying dorsal to the posterior commissure.
F.—*L. Col.*—Sclerosis of crossed pyramidal tract.
G.—*A. Col.*—Sclerosis of direct pyramidal tract, bordering on anterior fissure, but less intense than in the posterior columns.
H.—*P. Horn.* } Nerve cells less numerous than normal, and much altered.
J.—*A. Horn.* }
K.—*Clarke's Col.*—No mention.
L.—*Sp. Roots.*—Posterior sclerosed.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*
O.—*C. Canal.*
P.—*Med.*
Q.—*Cbl.*
R.—*Cbr.*
S.—*Remarks.*—Smith thinks the disease due to arrested development of some of the cells and fibres of the cord.

Case 7.

A.—*Duration.*—R. S.; duration 14 years. Died at 29 of cardiac dilatation and failure.
B.—*Author.*—Newton Pitt. *Guy's Hospital Reports*, 1887, p. 369.
C.—*Cord.*—Unusually small.
D.—*Memb.*—Unaffected.
E.—*P. Col.*—Sclerosed. Goll's column throughout. Burdach's column severely so in posterior part. Escape of a narrow strip bounding posterior horn and root, especially along anterior half.
F.—*L. Col.*—Sclerosis of crossed pyramidal and cerebellar tracts. Most marked in dorsal region; less so above. A strip of marginal sclerosis extends forward, anterior to the direct cerebellar tract (Gower's tract ?).
G.—*A. Col.*—Periphery slightly sclerosed above, extending, in lower cervical region, to antero-median fissure.
H.—*P. Horn.*—In some sections sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*
O.—*C. Canal.*
P.—*Med.*—Small.
Q.—*Cbl.*
R.—*Cbr.*—Normal.
S.—*Remarks.*—Rutimeyer considered the degeneration in both these cases to be identical with that occurring in tabes.

Case 10.

A.—*Duration.*—Paul Par.; duration 11 years. Died at 21, of mitral stenosis and pulmonary apoplexy.
B.—*Author.*—Déjérine and Letulle. *La Médecine Moderne*, April, 1890.
C.—*Cord.*—Small.
D.—*Memb.*—Adherent.
E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column. Marked sclerosis of Burdach's. Sclerosis of Lissauer's tract.
F.—*L. Col.*—Slight sclerosis of crossed pyramidal tract. Medium atrophy of direct cerebellar tract.
G.—*A. Col.*—Unaffected.
H.—*P. Horn.*—Shrunken in lumbar region.
J.—*A. Horn.*
K.—*Clarke's Col.*—Atrophy of fibres and cells.
L.—*Sp. Roots.*—Atrophy of posterior roots.
M.—*Sp. Ganglia.*
N.—*Peri. Nerves.*
O.—*C. Canal.*—Peri-ependymal glioma.
P.—*Med.*
Q.—*Cbl.*
R.—*Cbr.*
S.—*Remarks.* — Déjérine described in this case whorls of neuroglial fibres, which in L. S. appeared as undulating intercrossing fibres of great length. While this was the condition of the sclerosed posterior columns, in the lateral column on the other hand the sclerosis was a vascular one, and was associated with thickening of the pial prolongations. The lateral sclerosis he considered secondary to a meningo-myelitis.

Case 11.

A.—*Duration.*—Suzanne Desch.; duration 9 years. Died at 19, of pulmonary phthisis.
B.—*Author.*—Blocq and Marinesco. *Archives de Neurologie*, May, 1890.
C.—*Cord.*—Small.
D.—*Memb.*—Normal.
E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column. Severe sclerosis of Burdach's column. Escape of Westphal's postero-marginal zone. Lissauer's tract affected below but intact above.
F.—*L. Col.*—Sclerosis of crossed pyramidal and of direct cerebellar tracts, most marked in dorsal region. In lateral column, dilatation of

vessels, with symmetrically placed hæmorrhagic exudates. Thickened pial trabeculæ are present in the sclerosed area. Gower's tract unattacked.

G.—*A. Col.*—Unaffected.

H.—*P. Horn.*—Diminution in number of large transversal fibres. Ascending fibres less affected.

J.—*A. Horn.*

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Sclerosis of posterior roots. Most marked in lumbar region.

M.—*Sp. Ganglia.*—Attacked. Disappearance of fibres. Peripheral vacuolation of ganglion cells. Hypertrophy of connective tissue.

N.—*Peri. Nerves.*—Not examined.

O.—*C. Canal.*

P.—*Med.*—Loss of fibres from posterior pyramids, less marked in corpora restiformia. Anterior pyramids unaffected. Cerebellar tract degenerated. Above olive a cavity, apparently due to capillary dilatation. Destruction of fibres of raphe.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Blocq and Marinesco consider the lesions in all the columns affected to consist in atrophy of nerve fibres and thickening of neuroglia. The fibres present have no relation to the pial system. They consider the morbid change starts from blood vessels, but that there is also a developmental lesion.

Case 12.

A.—*Duration.*—A. G.; duration 18 years. Death at 28 from heart disease.

B.—*Author.*—Guizetti. *Riforma Medica*, June, 1893, and *Il Policlinico*, 1894, p. 438.

C.—*Cord.*—Small.

D.—*Memb.*

E.—*P. Col.*—Sclerosed; with exception of a tract posterior to commissure and adjacent to posterior cornua (cornu-commissural tract ?). Escape of Flechsig's "centrum ovale." Lissauer's tract degenerated in dorsal region. Degeneration less severe in cervical region, where there is improvement in Burdach's column.

F.—*L. Col.*—Many degenerated fibres in crossed pyramidal tract in lumbar and dorsal region. Fewer in cervical region. D. C. T., and Gower's tract attacked from dorsal region upwards. Escape of lateral limiting layer.

G.—*A. Col.*—Unaffected.

H.—*P. Horn.*—Atrophied, cells few and degenerated.

J.—*A. Horn.*—Normal.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots sclerosed. Anterior healthy.

M.—*Sp. Ganglia.*—Diminution in size of ganglion cells with progressive atrophy of some. Compensatory hyperplasia of connective tissue.

N.—*Peri. Nerves.*—Much atrophied. Many fine fibres present.

O.—*C. Canal.*—Pervious for a short distance only.

P.—*Med.*—Degeneration of Goll's and Burdach's columns. Horizontal fibres unaffected. Some degenerated fibres in anterior pyramids.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Guizetti found no alteration of pial septa, or of blood vessels. The fine fibres in the peripheral nerves proceed solely from the posterior roots, and are therefore sensory. The fine fibres in the muscular branches have the same origin. Guizetti concluded that in this case of Friedreich's disease there existed a systematic atrophy of sensory nerves, both cutaneous and muscular.

Case 13.

A.—*Duration.*—L. P.; duration 5 years. Death at 19 from tubercular pleurisy.

B.—*Author.*—Mirto. *Giornale del'Assoc. dei Medici e Naturalisti*, Anno iv., 1893.

C.—*Cord.*

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Goll's and Burdach's columns degenerated. Lissauer's tract attacked.

F.—*L. Col.*—Degeneration of crossed pyramidal tract, diminishing from below upwards. Slight degeneration of direct cerebellar tract, and of Gower's tract.

G.—*A. Col.*

H.—*P. Horn.* }
J.—*A. Horn.* } Thinning of nervous reticulum in anterior and posterior horns, with atrophy of cells. Most marked in dorsal region.

K.—*Clarke's Col.*—Extreme sclerosis.

L.—*Sp. Roots.*—Posteror roots sclerosed.

M.—*Sp. Ganglia.*—Atrophic degeneration of cells. Thinning of nervous reticulum. Hyperplasia of connective tissue.

N.—*Peri. Nerves.*—Motor nerves degenerated; sensory not examined.

O.—*C. Canal.*

P.—*Med.*

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The sympathetic ganglia were also examined and found normal.

Case 14.

A.—*Duration.*—Female; duration 18 years. Died at 28 from diabetes and tuberculosis.

B.—*Author.*—Burr. *University Medical Magazine*, Philadelphia, June, 1894.

C.—*Cord.*

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Extreme sclerosis of Goll's column; less severe of Burdach's; from lower lumbar to highest cervical region.

F.—*L. Col.*—Degeneration of C. P. T. from lumbar swelling to upper cervical region. Degeneration of D. C. T. from mid-dorsal to upper cervical.

G.—*A. Col.*—Degeneration of D. P. T. from mid-dorsal to upper cervical regions.

H.—*P. Horn.*—Slight affection of posterior horns. Degeneration of ganglion cells.

J.—*A. Horn.*—Atrophy of a few cells.

K.—*Clarke's Col.*—Marked degeneration.

L.—*Sp. Roots.*—Posterior sclerosed; anterior intact.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—The reporter adheres to Dejerine and Letulle's theory of the nature of the changes in the posterior and lateral columns.

Case 15.

A.—*Duration.*—Male; duration 8 years. Died at 19.

B.—*Author.*—Dana. *Postgraduate*, New York, vol. xi., no. 7.

C.—*Cord.*—Small; flattened antero-posteriorly.

D.—*Memb.*—Pia thickened.

E.—*P. Col.*—Sclerosed most at lower levels.

F.—*L. Col.*—Sclerosed. Affecting C. P. T. and D. C. T., and to a more or less extent, Gower's tract. In addition, marginal sclerosis of nearly the whole circumference of the cord is present.

G.—*A. Col.*

H.—*P. Horn.*—Moderate degree of degeneration of grey matter affecting nerve cells.

J.—*A. Horn.*

K.—*Clarke's Col.*

L.—*Sp. Roots.*—Not examined.

M.—*Sp. Ganglia.*—Not examined.

N.—*Peri. Nerves.*—Not examined.

O.—*C. Canal.*

P.—*Med.*—Degeneration "probably" extended into medulla.

Q.—*Cbl.*

R.—*Cbr.*

S.—*Remarks.*—For clinical history see *Journal of Mental and Nervous Diseases*, vol. xv., 1890. Numerous holes (? perivascular spaces) ·5 to 2 mm. in size were scattered through grey and white matter of the cord. Presence of gliosis in sclerosed area could not be affirmed.

Case 16.

A.—*Duration.*—Ad. Rouzier; duration 5 years. Death at 17 from unexplained cause, not revealed on autopsy.

B.—*Author.*—Simon. *Le Progrès Médical*, September, 1897.

C.—*Cord.*—Small.

D.—*Memb.*—Thickening of soft membranes on posterior aspect of cord.

E.—*P. Col.*—Sclerosed. Most marked in dorsal region, but almost as

severely in cervical, too. Lissauer's tract attacked in sacral region, but cornu-commissural and septo-marginal fibres escape. In lumbar region, Flechsig's centrum ovale is attacked, but cornu-commissural fibres again escape.

F.—*L. Col.*—Sclerosed, but less so than posterior column. The C. P. T. and D. C. T. equally affected from the upper lumbar region upwards. The lateral limiting layer escapes.

G.—*A. Col.*—Direct pyramidal tract affected from upper dorsal region upwards.

H.—*P. Horn.*—Myelinic reticulum degenerated. Diminution in number and in volume of ganglion cells.

J.—*A. Horn.*—Diminution in number and volume of ganglion cells, but the motor cells of front part of the cornua are well developed.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots sclerosed throughout; more so in lumbar and dorsal regions, less so in cervical.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*

P.—*Med.*—Sclerosis of cuneate nucleus. More severe sclerosis of funiculus gracilis. Cerebellar bundle attacked.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The neuroglial tissue in the sclerosed areas is in some places arranged in perivascular whorls. Vessels but slightly altered. No marked proliferation of interstitial nuclei.

Case 17.

A.—*Duration.*—Jules G.; duration 13 years. Died at 39 of pleurisy (tubercular).

B.—*Author.*—Bonnus. *Nouvelle Iconographie de la Salpétrière*, No. 3. 1898.

C.—*Cord.*—Small.

D.—*Memb.*—Healthy.

E.—*P. Col.*—Sclerosed. Most marked in dorsal region. Posterior and median radicular zones most attacked. Anterior radicular only affected above dorsal region. Lissauer's tract attacked in sacral and lumbar regions; less so in dorsal. Cornu-commissural tract and centrum ovale relatively, but not absolutely, preserved.

F.—*L. Col.*—Sclerosed, but less so than the posterior column. C. P. T. attacked throughout. D. C. T. attacked at and above dorsal region. Gower's tract also attacked.

G.—*A. Col.*—Direct pyramidal tract sclerosed in dorsal region, and still more affected in cervical region.

H.—*P. Horn.*

J.—*A. Horn.*

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior roots much degenerated.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Median, sciatic, anterior tibial, and musculo-cutaneous degenerated.

O.—*C. Canal.*

P.—*Med.*—Goll's and Burdach's nuclei attacked; otherwise normal.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Bonnus's case was diagnosed as Friedreich's disease by Charcot. It was characterised by lightning pains, which have been recorded in Friedreich's disease by Charcot and Dejerine.

Case 18.

A.—*Duration.*—Henry W.; duration 24 years. Died at 30, of heart failure during measles.

B.—*Author.*—Mackay.

C.—*Cord.*—Small.

D.—*Memb.*—Healthy; some thickening of dura.

E.—*P. Col.*—Sclerosed. Maximum degree in upper dorsal. Least degree in lowest lumbar, where cornu-commissural tract escapes relatively. Some fibres of septo-marginal tract escape in mid-lumbar. Lissauer's tract sclerosed, but gradual improvement in same from upper dorsal region upwards. Posterior root zones improve *pari passu* with Lissauer's tract.

F.—*L. Col.*—Sclerosed. C. P. T. from lumbar enlargement to upper cervical. D. C. T. from lower dorsal to calamus scriptorius. Maximum degeneration in mid-dorsal region. Lateral limiting layer intact throughout. Gower's tract slightly attacked in cervical region.

G.—*A. Col.*—Direct pyramidal tracts on both sides sclerosed from lower dorsal upwards. Maximum in cervical region.

H.—*P. Horn.*—Sclerosed both in longitudinal and transverse fibres. Ganglion cells for most part absent. Fibrillar plexus poor in myelin.

J.—*A. Horn.*—Normal, except for some slight diminution in volume of motor cells.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Posterior sclerosed, but contain many fine fibres. Anterior show slight degenerative change.

M.—*Sp. Ganglia.*—Shrinkage and extreme pigmentation of ganglion cells. Increase of interstitial connective tissue. Degeneration of ascending fibres passing through ganglia.

N.—*Peri. Nerves.*—Sciatic, anterior crural, anterior tibial, internal plantar, median ulnar, radial and posterior interosseous, all extremely degenerated.

O.—*C. Canal.*—Occluded by round cells in most of its course. Normal above decussation of pyramids.

P.—*Med.*—Sclerosis of cerebellar tract. Sclerosis of funiculi graciles and cuneati. Some degeneration of pyramidal tracts and of Gower's tracts. Improvement of all parts after passing upper level of olives.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Vessels of cord healthy and distended with blood-cells, but no exudates. No perivascular whorls in glial tissue. Abnormalities, probably gliomatous, present in dorsal and cervical regions of cord.

SUMMARY OF AUTOPSIES ON DOUBTFUL CASES.

Case 1.

A.—*Duration.*—Strasik Josepha; duration 7 years. Death at 23. Mitral disease, and probably tuberculosis present.

B.—*Author.*—Kahler and Pick. *Archiv für Psychiatrie*, Bd. viii., s. 251.

C.—*Cord.*—Small.

D.—*Memb.*—Adhesion of dura and pia.

E.—*P. Col.*—Degenerated throughout, except a thin zone next grey matter.

F.—*L. Col.*—Posterior parts degenerated up to periphery. Lateral limiting layer unattacked. Crossed pyramidal tract more affected on right side than on left. Direct cerebellar tract degenerated.

G.—*A. Col.*—Right side degenerated down to dorsal region; left unaffected. (The reporters suppose there was an anomalous arrangement of decussating fibres).

H.—*P. Horn.* } No change in grey matter beyond a relatively feeble
J.—*A. Horn.* } development.

K.—*Clarke's Col.*—Sclerosed.

L.—*Sp. Roots.*—Anterior normal. Posterior sclerosed.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves*

O.—*C. Canal.*

P.—*Med.*—Normal.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*

Case 2.

A.—*Duration.*—Marie R.; duration 8 years. Died at 32 of phthisis.

B.—*Author.*—Brousse. *De l'ataxie héréditaire*, Paris, 1882.

C.—*Cord.*—No mention of size.

D.—*Memb.*

E.—*P. Col.*—Sclerosed. Most marked in lumbar region, and least in dorsal.

F.—*L. Col.*—Posterior parts sclerosed but not up to the periphery. Most marked in cervical and lumbar regions.

G.—*A. Col.*—Turck's column unaffected, but sclerosis present on both sides around heads of anterior cornua. Most extensive on left side.

H.—*P. Horn.*

J.—*A. Horn.*—Tips of cornua slightly affected in cervical region.

K.—*Clarke's Col.*—Not mentioned.

L.—*Sp. Roots.*—Not mentioned.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*

O.—*C. Canal.*—"Inflamed." Lumen diminished by a collection of small embryonal cells. Complete obstruction in lumbar region.

P.—*Med.*—Slight sclerosis in posterior pyramids.

Q.—*Cbl.*—Normal.

R.—*Cbr.*—Normal.

S.—*Remarks.*—Apoplectiform attacks present in this case.

Case 3.

A.—*Duration.*—N. N., female; duration 4½ years. Died at 29 of pulmonary phthisis.

B.—*Author.*—Auscher. *Archives de Neurologie*, 1890, p. 475, and *Semaine Medicale*, July, 1890.

C.—*Cord.*—Small.

D.—*Memb.*—Normal.

E.—*P. Col.*—Sclerosed. (A neuroglial sclerosis with integrity of the vessels and pial prolongations.) Lissauer's zone intact.

F.—*L. Col.*—Unaffected.

G.—*A. Col.*—Unaffected.

H.— *P. Horn.*—Atrophied.

J.—*A. Horn.*

K.—*Clarke's Col.*—Atrophy of fibres and diminution of cells.

L.—*Sp. Roots.*—Posterior roots contain many "embryonal" fibres, but no empty sheaths.

M.—*Sp. Ganglia.*

N.—*Peri. Nerves.*—Sensory nerves show no degeneration of tubes or empty sheaths, but a great number of fibres with embryonal characters.

O.—*C. Canal.*—Replaced by a mass of epithelial cells.

P.—*Med.*

Q.—*Cbl.*—Small, but not degenerated.

R.—*Cbr.*—Small.

S.—*Remarks.*

Case 4.

A.—*Duration.*—Eg. Pall.; duration 6 years. Death at 18 from chronic pneumonia followed by marasmus.

B.—*Author.*—Tedeschi. *Beiträge zur Pathologischen Anat. und Allgemein. Pathol.*, Bd. xx., Hft. 1, 1896.

C.—*Cord.*—Small.

D.—*Memb.*—Dura normal. Pia thickened in posterior part.

E.—*P. Col.*—Sclerosed. Degeneration extending out to middle root zone. Escape of cornu commissural zone, and to a less extent of postero-internal root zone. Lissauer's tract attacked.

F.—*L. Col.*—Degenerated fibres in cervical region in area of D. C. T. At other levels merely abnormal thinness of nerve fibres.

G.—*A. Col.*—Abnormal thinness of nerve fibres.

H.—*P. Horn.*
J.—*A. Horn.*
} Thinning of nervous reticulum. Diminution and pigmentation of cells. Those of posterior horn shrunken and deformed.

K.—*Clarke's Col.*—Degeneration of cells. Thinning of fibrillar plexus.

L.—*Sp. Roots.*—Atrophied. The various fasciculi composed of extremely fine fibres which react very feebly with specific stains. Interstitial tissue increased.

M.—*Sp. Ganglia.*—Striking thinness of nerve fibres. Axis cylinders and myelin sheaths stain badly with their proper stains. Some cells vacuolated, some over-pigmented. Interstitial tissue increased.

N.—*Peri. Nerves.*—Not examined.

O.—*C. Canal.*—Double in lumbar region, becoming single at higher level.

P.—*Med.*—Sclerosis of Goll's and Burdach's funiculi. Longitudinal fibres diminished. Horizontal ones diminished and thinned. Cells less

numerous, shrunken, and pigmented. Reil's band appears unaltered both at origin and crossing. Pyramids, arciform fibres, and crura cerebri normal.

Q.—*Cbl.*—Normal size. Increase of pigment in Purkinje's cells. Thinning of fine plexus in nuclear layer.

R.—*Cbr.*—Normal.

S.—*Remarks.*—The patient was insane.

LITERATURE.

Autopsies on Friedreich's disease, including doubtful cases of the same.

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[2] SCHULTZE, *Virchow's Archiv*, Bd. lxxix, s. 132.
[3] KAHLER AND PICK, *Archiv für Psychiatrie*, Bd. viii., s. 251.
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[5] EVERETT SMITH, *Boston Medical and Surgical Journal*, 1885.
[6] NEWTON PITT, *Guy's Hospital Reports*, 1887, p. 369.
[7] RUTIMEYER, *Virchow's Archiv*, Bd. cx., s. 215.
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[19] MENZEL, *Archiv für Psychiatrie*, Bd. xxii., s. 160. (Case of cerebellar atrophy, with cord changes similar to those of Friedreich's disease).
[20] FRASER, *Glasgow Medical Journal*, 1880. (Cerebellar atrophy, with normal condition of cord.)
[21] MEYER, BRAIN, part lxxix., vol. xx. (Autopsy on one of Sanger Brown's series. No circumscribed lesion in cerebellum. Degeneration of posterior columns, and of D. C. T. in cervical cord.)

The following have also been referred to:—

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[24] GUDDEN, *Archiv für Psychiatrie*, Bd. xxviii., s. 643. (Changes in peripheral nerves in neuritis. With summary of literature on segmental neuritis and regeneration processes.)
[25] WESTPHAL, *Archiv für Psychiatrie*, Bd. viii., xiv., and xvi.
[26] SAKAKY, *Archiv für Psychiatrie*, Bd. xv., s. 584.
[27] NONNE, *Archiv für Psychiatrie*, Bd. xix., s. 352.
[28] OPPENHEIM AND SIEMERLING, *Archiv. für Psychiatrie*, Bd. xviii., s. 98.
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DESCRIPTION OF PLATES.

(Staining in each case by Schäfer's method. Figs. 1-5 from microphotograph and sketch. Figs. 6 to 8 microphotograph only. Figs. 1-5, × 10 diameters. Fig. 6, × 8 diameters. Figs. 7 and 8, × 70).

FIG. 1.—T. S. at L. 4. Sclerosis of posterior column, except in anterior fourth. Also of posterior roots, root-zones, and cornua. Escape of fibres in position of Flechsig's *centrum ovale*. Slight degeneration of C. P. T. on each side.

FIG. 2.—T. S. at L. 1. Sclerosis of posterior columns, with exception of fibres in position of cornu-commissural tract. Also of posterior roots, root-zones, and cornua. Sclerosis of Clarke's Columns. Degeneration of C. P. T.

FIG. 3.—T. S. at D. 6. Structural abnormality in posterior column (developmental ?).

(*a*) Semilunar area of sclerotic tissue, surrounded by a whorl of degenerate myelinised fibres.

(*b*) Capsule of sclerotic tissue enclosing a central medullated (degenerated) area, and in its turn enclosed by a layer of degenerated medullated fibres.

These latter probably represent those previously occupying the ventral fields of the posterior column, and bounding the posterior cornua.

FIG. 4.—T. S. at D. 2. Sclerosis of posterior column here reaches its maximum. Commencing improvement in root fibres, and in postero-external fields of Burdach's column. Atrophy of posterior cornua and Clarke's columns. Sclerosis of C. P. T., D. C. T. and D. P. T.

FIG. 5.—T. S. at C. 5. Abnormality in left posterior cornu. Structure somewhat similar to that described in fig. 3. Sclerosis of Goll's column. Improvement in postero-external fields of Burdach's columns. Well-stained fibres adjoining posterior cornua. Sclerosis of C. P. T., D. C. T. and D. P. T.

FIG. 6.—T. S. Medulla at decussation of fillet layers.

(*a*) Funiculus gracilis, and nucleus, sclerosed.

(*b*) Funiculus cuneatus, partially sclerosed, with poorly developed nucleus.

(*c*) Substantia gelatinosa. The fibrillar reticulum of the posterior cornu is fairly well myelinised.

(*d*) Direct cerebellar fibres, sclerosed.

(*e*) Gower's tract, slightly attacked.

(*f*) Fillet layers. Well-stained and healthy.

(*g*) Decussation of internal arcuate fibres. Their further course from posterior horn and Burdach's nucleus does not appear well in this section.

(*h*) Asymmetry and partial degeneration of anterior pyramids.

FIG. 7.—T. S. Sciatic. Schäfer. × 70.

FIG. 8.—T. S. Median. Schäfer. × 70.

A CASE OF TUMOUR OF THE PONS.

BY J. DIXON MANN, M.D., F.R.C.P.
Physician to the Salford Royal Hospital,
AND
SHERIDAN DELÉPINE, M.B., B.Sc.
Professor of Pathology in The Owens College.

WILLIAM S., aged 52 years, was admitted into the Salford Royal Hospital on August 11, 1893. He enjoyed good health until March, 1892, when he began to have shooting pains in the back of the head; the pains were subsequently felt at the top and the front of the head. He vomited occasionally. In December, 1892, he began to have humming noises in the left ear. In April, 1893, he felt a pain in the right shoulder, which extended down the arm to the hand. He has muscular twitches in the left leg and he feels rather dizzy. After remaining in hospital for several weeks, he was discharged at his own request, but he continued to attend as an out-patient. The right arm and right leg now began to feel weak, and in September, 1894, he had ptosis of the left eye, which lasted three months. In the early part of December he had a succession of convulsive attacks. The noise in the left ear has been continuous.

On February 4, 1895, he was re-admitted, when his condition was as follows:—The right arm, flexed at a right angle, was spastically contracted and devoid of motor power. The right leg was also spastic and devoid of motor power. There was no paralysis of the facial muscles and the ptosis had disappeared —the left eye could be freely opened. There was no perceptible paralysis of the ocular muscles—the eye ball was capable of the usual movements, nor was there any nystagmus. The pupils were equal, not contracted, and they reacted to light. There was double optic neuritis and slight limitation of the field of

vision on the temporal side of the left eye; vision was otherwise unaffected. The humming or buzzing sound in the left ear was now associated with impaired audition on the same side. The perception of acid substances applied to the tongue was equally good on both sides. The pain in the head had almost disappeared. When tested with the point of a pin and with test-tubes respectively filled with hot and cold water, sensation was found to be unaffected on both sides of the face, on the arms and on the legs. On the right side the knee-jerk was slightly increased. The speech was much impaired, being thick and "bulbar"; there was also some difficulty in swallowing. The mental faculties were unaffected.

Whilst in hospital the patient had twenty to thirty seizures, in which, for the most part, the head and the eyes were turned to the right side, the left eye being less deflected than the right. When the patient was in a passive condition, no difference was perceptible between the movements of the left eye and those of the right. The seizures were characterised by a condition of tonic spasm, which lasted from a few seconds up to two or three minutes, the right arm and the right leg being absolutely stiff and rigid. The left arm and leg were not affected. The pupils were dilated and did not respond to light. The respirations were slowed. During the seizures and for a varying period afterwards—from a few minutes to half an hour—the patient was quite unconscious. On one or two occasions there were slight indications of an epileptiform character, and once, slow clonic spasms—one every four or five seconds—were observed to affect the right arm. In two of the seizures the right side of the face appeared to be partially paralysed, the cheek "flapping" with the respirations at the maximum of the attack. In February, 1895, both sides of the face, both arms, and both legs twitched during an attack. On another occasion the twitching was limited to the fingers of the right hand, the face not being affected. On March 21 the left eye was observed to remain closed for about twelve hours after an attack. During the interval between the attacks, if the patient (who was then quite conscious and intelligent) was asked a question, the answer to which involved some reflection, the left eyelids usually twitched convulsively. After a seizure on March 27, ptosis on the left side recurred and persisted until death, which took place on April 4, 1895. No sugar was found in the urine, nor albumin until a fortnight before death.

Professor Delépine's Report on the brain, cerebellum, pons and medulla oblongata.

These parts having been kept in weak spirit for a few days before being available for examination were very soft, and had partly lost their original colour. The only evidences of disease which could be detected by an external examination were the following:—

(1) A marked swelling of the left side of the pons, most distinct above the exit of the 5th nerve, and causing much asymmetry of the pons.

(2) A very considerable enlargement of the left peduncle of the brain.

(3) Great displacement of the basilar, left posterior cerebral and left superior cerebellar arteries as well as of the left 3rd and 4th nerves, and to a lesser degree of the 5th nerve.

(4) Atrophy of the left 3rd nerve, which measured about 2 mm. in diameter, whilst the right 3rd measured over 3 mm. in diameter.

(5) On separating slightly the left tempero sphenoidal lobe from the left crus, a tumour became apparent. Owing to the soft state of the lateral portion of the left crus, and of consequent lacerations of this part, the superficial relations of this tumour could not be very clearly ascertained. It was, however, evident that the tumour, which seemed to be of the size of a pigeon's egg, must have compressed the posterior parts of the left optic tract.

(6) When the upper surface of the cerebellum was exposed, a distinct bulging was evident in the anterior part of the left quadrate lobe. This swelling was situated about $\frac{1}{2}$ inch to the left of the superior vermiform process, which was displaced towards the right, so that, seen from above, the two halves of the cerebellum seemed unequal. The left cerebellar hemisphere looked larger than the right.

Owing to the soft state of the specimen, it was not found possible to make many sections through the parts affected. The following were, however, found sufficient to determine with tolerable accuracy the localisation of the

DIAGRAM I.

Diagram of left aspect of medulla, pons, crus cerebri, thalamus opticus, etc., to show the relations of the tumour and surrounding areas of degeneration as well as the direction of the various sections described in the text.

1 to 6.—Planes of sections. *II. to VIII.—Cranial nerves.*

A—Pulvinar.
B—External and internal geniculate bodies.
C—Pineal gland.
D—Corpora quadrigemina.
E—Peduncle of cerebrum.
F—Left corpus albicans.
G—Superior peduncle of cerebellum.
H—Middle „ „ „
K—Inferior „ „ „
L—Medullary centre of left hemisphere of cerebellum. The letter is placed at the confluence of the primary medullary lamellæ of the anterior part of the antero-superior lobule and of the lateral part of the central lobule.
M—Olive.

The tumour is indicated in black, and the degeneration by light shading.

DIAGRAM II.

Section 1 (seen from behind).

A—Internal capsule.
B—Substantia nigra.
C—Optic thalamus.
*C**—Left optic thalamus at the place where degeneration has extended from the tegmental portion of the peduncle (degenerated part indicated by the darker shade).
D—Lenticular nucleus.
E—Velum interpositum.
F—Fornix.
G—Corpus callosum.

This sketch should be reversed to make it comparable with the other drawings.

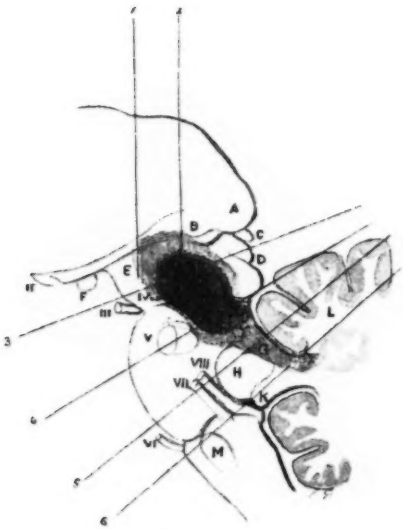


Diagram I.

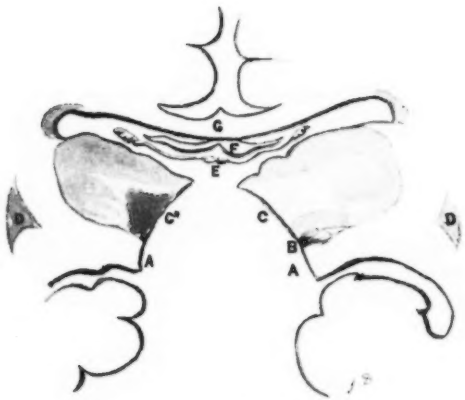


Diagram II.

lesions, except in the left external part of the left crus, which had been destroyed by softening and *post-mortem* laceration.

The planes of section will be best understood by reference to the diagram I., which represents the posterior parts of the basal ganglia, crura cerebri, pons varolii, medulla, cerebellum, and peduncle of the cerebellum, seen from the left side. In this diagram, the affected parts are shaded, the tumour itself deeply, the softened parts less deeply. All these parts could not be seen from the surface, as has already been explained.

Section 1 (Diagram II.).—A vertical frontal section passing through the hemispheres of the brain and dividing the basal ganglia behind the grey commissure. It shows the relations of the tegmental and crustal portions of the cerebral peduncles to the region of the optic thalami and the internal capsula. A small portion of the locus niger indicates the line of demarcation between the tegmentum and crusta. A patch of degeneration is seen to have destroyed a small portion of the left optic thalamus and subthalamic region. The internal capsule is apparently unaffected.

Section 3 (Diagram III.).—This section was slightly oblique, dividing the crura just above the superior corpora quadrigemina, and the point of exit of the 3rd nerves.

A very vascular tumour, surrounded by a layer of very soft, degenerated, nervous tissue, occupies the space between the grey matter of the corpora quadrigemina posteriorly, and the crusta anteriorly, the mesian plane on the right, and the surface of the peduncle on the left.

It is at this level that part of the tumour and surrounding tissues had been accidentally destroyed, but the shape and relations of the tumour indicate that it had probably extended from the surface of the crus, and originated in the meningeal covering. There can be little doubt either that the left corpora geniculata were involved. The left crusta, except in its most external part (temporo-occipital cerebellar fibres and pyramidal fibres for the leg), was not involved, but must have been subjected at times to considerable pressure. The grey matter round the aqueduct had been simply dis-

Diagram III.

Section 3.—Passing through the superior corpora quadrigemina, the aqueduct, the crusta, a little above point of exit of the third nerves (seen from above).

A—Superior corpora quadrigemina.
B—Brachium of left testis.
C—Crusta; letter placed opposite pyramidal fibres.
D—Substantia nigra.
E—Aqueduct.
F—Right third nerve.
F'—Left third nerve.
The relative size of the two nerves is represented by means of transverse sections, below the main diagram.
G—Basilar artery with its branches.
H—Tumour (dark shade).
I—Zone of softening round the tumour (lighter shade).

Diagram IV.

Section 4.—Passing through pons Varolii at level of exit of fifth nerve, upper part of superior cerebellar peduncles just below the inferior corpora quadrigemina, inferior corpora quadrigemina, and most anterior and superior cerebellar laminæ (seen from above).

A—Superior cerebellar peduncle.
B—Fifth nerve.
C—Pyramidal bundles.
D—Superficial transverse fibres of pons.
E—Deep transverse fibres of pons.
F—Fillet.
G—Aqueduct near upper end of fourth ventricle.
H—Anterior end of superior vermiform process.

Tumour and degenerated parts indicated as in previous diagrams.

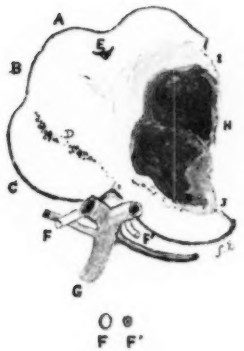


Diagram III.

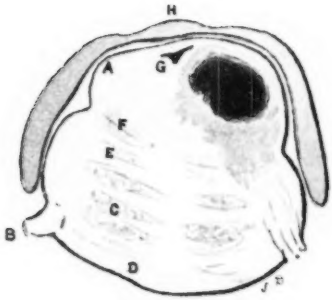


Diagram IV.

placed towards the right. It seems impossible that the fibres of the left 3rd nerve should have escaped destruction in their passage from their nucleus to the surface, and as a matter of fact the left 3rd was in a state of advanced atrophy.

Section 4 (Diagram IV.).—This section is slightly more oblique than the last, and followed the fibres of the 5th nerve for some distance through the pons.

In this section the lower part of the tumour is seen to extend beneath the fibres of the superior cerebellar peduncle, which seem to be partly destroyed by it. The zone of softening round the tumour extends more in the direction of the fibres of the 5th than in any other direction. The aqueduct is pushed towards the right, and part of the grey matter round it on the left side remains apparently unaffected. The right half of the tegmental and ventral parts of the pons are but slightly affected by pressure at this level, but the left half is still considerably enlarged.

Section 5 (Diagram V.).—This section is a little more oblique than the previous one. It is entirely below the level of the tumour, and shows only a tract of degenerated tissue in the region corresponding to the superior cerebellar peduncle and adjacent white matter. Though the left half of the pons is distinctly larger than the right, it seems almost entirely free from lesion.

Section 6 (Diagram VI.) is nearly parallel to the last. It passes through the upper part of the medulla oblongata, showing the exit of the 8th nerve; it also divides the floor of the 4th ventricle at about the level of the striæ acusticæ, it exposes the white centres of the cerebellum, with a small part of the corpora dentata; the relations of the central lobule are also well shown.

The only lesion visible at this level is a small patch of softening evidently following the direction of the fibres of the superior cerebellar peduncle and involving the region of the left corpus dentatum. There is a slight displacement of the central lobule towards the right; the floor of the 4th ventricle may have been at times compressed by the central lobule when the diseased parts underwent rapid enlargement from one cause or another.

DIAGRAM V.

Section 5.—Passing through the pons Varolii at a level half way between lower edge of the pons and exit of the fifth nerves (seen from above).

A—Superior medullary velum, behind fourth ventricle.
B—Middle peduncle of cerebellum.
C—Seventh and eighth nerves.
D—Pyramidal fibres.
E—Flocculus.
F—Superior or quadrate lobe.
G—Vermiform process.

Softened part shaded as in previous diagrams.

DIAGRAM VI.

Section 6.—Passing through medulla immediately below lower border of pons, through the central lobule and postero-superior lobule of cerebellum (seen from above).

A—Anterior pyramid.
B—Root of eighth nerve.
C—Central lobule.
D—Upper part of right corpus dentatum.

The patch of softening is indicated as in other diagrams.

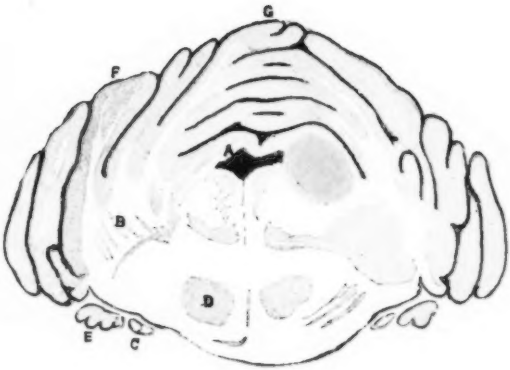


Diagram V.

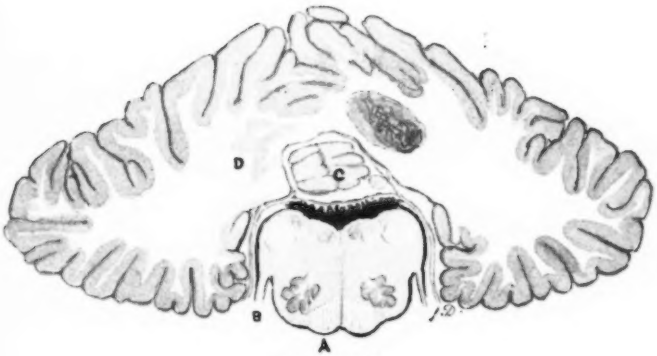


Diagram VI.

From a study of these sections one may safely come to the following conclusions :—

The left crus was the seat of a tumour which had destroyed the greater part of its tegmentum. This tumour had apparently originated near the external left aspect of the crus, probably extending from the membranes and inserting itself like a wedge between the left corpora quadrigemina and the crustal portion of the left cerebral peduncle, causing them to become widely separated and disconnected. This tumour did not extend upwards higher than the upper parts of the upper corpora quadrigemina, and downwards below the upper border of the middle cerebellar peduncle; the formatio reticularis and the red nucleus were the parts primarily invaded and destroyed by the tumour (within the region affected); other parts were also destroyed, *more or less* completely, being involved in the zone of degenerated and necrosed tissues surrounding the tumour; these were the left superior cerebellar peduncle, the left upper and lower fillet, the left corpora geniculata and posterior part of the left optic tract, the fibres of the root of the left third nerve; the left 4th nerve was possibly involved, in its course round the diseased crus. Many of the fibres of the left 5th must also have been involved.

The nuclei of the 3rd, 4th, 5th, were all in close contact with the tumour, and partly involved in the necrosed zone.

The fibres of the 2nd, 3rd, 4th, 5th, 6th, 7th, 8th nerves had either to pass through degenerated zones or were subjected to pressure, which must have been very variable in degree, considering the very vascular nature of the tumour. The external $\frac{1}{4}$th of the left crusta was partly infiltrated by the tumour, and had certainly much suffered from pressure; the rest of the left crusta was also subjected to pressure, though in a lesser degree. The pressure must also have at times affected indirectly the right half of the pons and the right crus.

The left 3rd nerve was the only nerve which showed striking evidences of the destructive action of the tumour.

This nerve was much atrophied; its diameter was only $\frac{3}{4}$rds of that of the corresponding nerve on the right side.

On microscopical examination not more than $\frac{1}{4}$th of its fibres could be recognised, and most of these were in a state of degeneration. Connective tissue occupied the space originally occupied by the other fibres. The right third was also partly degenerated, not more than $\frac{2}{3}$rds or $\frac{3}{4}$ths of its fibres presenting a normal appearance.

The tumour, by its situation, its relations to the formatio reticularis, the apparent hypertrophy of the pons produced by it, might have been supposed to be a typical glioma; but it was quite clearly a round-celled sarcoma. It was well defined, surrounded by a well marked zone of necrosed tissue, so soft that it was difficult to prevent a displacement of the parts. The tumour was nodulated, the nodules being firmly connected together by the condensed tissue intervening between them. The nodules were soft, very vascular, and owing to their dark colour, contrasting sharply with the surrounding opaque, yellowish white, surrounding necrosed nerve tissue. There was enough of this softened tissue to reconstitute the parts of the crus which seemed to have been replaced by the tumour, so that it is very doubtful whether there was any organic connection between the new growth and any part of the tegmentum. The cells of the tumour were small, rounded, with large nuclei, not separated by any distinct stroma except quite at the periphery of the nodules, where, through pressure, the cells were arranged in rows or packets separated by a very small amount of a clear, slightly fibrillated matrix.

The vessels were large, most of them with indistinct walls composed of embryonic looking cells; in some places the vessels were surrounded by several concentric layers of these cells. Though the tumour was almost angiomatous in a few places, there were but few traces of hæmorrhage; there was little evidence of degeneration in the central parts of the nodules. In all these respects the tumour resembled closely similar sarcomata growing from the soft membranes of the brain.

I therefore come to the conclusion that the case is one of round-celled sarcoma (very vascular) growing from the pia mater and penetrating into the left peduncle of the

brain, causing destruction of the tegmentum at that level, pressure on surrounding parts, and degeneration of certain fibres.

On reading Professor Delépine's description it is a matter for surprise that so many of the cranial nerves escaped suffering from the effects of the lesion; for not only were most of them directly involved by the tumour, but many had also to pass through a zone of degeneration and were subjected to varying pressure due to great vascular irregularity of the degenerated area. The third was the only nerve which showed definite atrophy, the paralytic symptoms being very partial; ptosis occurred, but no external strabismus, nor any alteration in the size or mobility of the pupil, nor in the power of accommodation. Lesions of the third nerve, however, often produce only partial results. No facial pain nor anæsthesia occurred from first to last, although the fifth nerve and its nucleus came well within the influence of the tumour; the seventh nerve also escaped, notwithstanding its dangerous contiguity. The slight indications of paralysis of the right side of the face on two occasions were due to diffuse pressure from increased vascularity. On the other hand, subjective auditory sensations were among the first and the most persistent of the symptoms; this, in a degree, is common, as are also spasm and convulsions in chronic lesions of the pons, the very gradual compression of the fibres of the pyramidal tract rarely causing sufficient irritation as to evoke spasm. The extremely slow growth of the tumour—three years and one month from the earliest symptom—probably accounts for the extraordinary tolerance displayed to its disturbing influence by the cranial nerves.

A FORM OF DISEASE RESEMBLING THE PSEUDO-SCLEROSIS OF WESTPHAL AND STRÜMPELL.

BY WILLIAM G. SPILLER, M.D.

Professor of Diseases of the Nervous System in the Philadelphia Polyclinic; Associate in the William Pepper Clinical Laboratory, University of Pennsylvania.

From the William Pepper Clinical Laboratory.

So few cases of this peculiar disease, described by Westphal and Strümpell, have been reported, that the following case, observed at the Pennsylvania Training School for Feeble-Minded Children, seems to be worthy of some attention.

T. H. M., male, was born in March, 1882, and died May 21, 1897. His parents were Irish, and his father was a labourer. Both parents were in good health prior to the birth of this boy. The mother died after the birth of her third child; according to the report, from abscess. She was subject to violent headaches. The father had a feeble-minded brother and an insane sister.

T. H. M.'s elder brother died in convulsions during an attack of scarlet fever. T. was the second child in the family, and was born at full term in ordinary labour, and was nourished by the breast. His mother was 39 years old at the time of his birth.

Nothing peculiar was noticed in the boy until he went to school. At this period his mental condition was found to be below the average. He did not defend himself from the attacks of his comrades, and was tormented by other boys in the street. He was said to have a good memory for objects, as well as considerable imagination, and he was able to relate stories. His gait and speech were rather slow, and he had a slight lisp. His circulation was poor, and his hands, as a rule, cold.

At the time of admission to the Pennsylvania Training School for Feeble-Minded Children, March 16, 1893, he was of good

size, with no striking peculiarities in the formation of the head. His sight and hearing were good. He was right-handed. No peculiar movements of face or body were noted. He did not laugh or weep without cause. He had no convulsions. He masticated his food well, was not gluttonous, and had fair table manners. He was quite active, not especially noisy, affectionate, and obeyed commands. He was trustful and truthful, tidy about his dress, and able to understand what was said to him.

April, 1893.—The boy could read quite readily at sight, and could count, but he had no knowledge of addition, and wrote a peculiar hand. He seemed interested in all the exercises the children were taught, though he at times appeared to be troubled because he could not always understand.

November, 1894.—After a long illness he re-entered school. He was retrograding, and seemed to have difficulty in going up and down stairs.

February, 1895.—Examination at this date showed impaired co-ordination, poor station, unequal pupils, and poor circulation. He had been gradually losing the use of his limbs.

December, 1896.—He had previously been able to sit in a chair, but could no longer do so, and fell forward when he attempted this. He had been ill several times. His legs were much atrophied, rigidity of all the joints was noted, and the hands and arms were so rigid and atrophied that he was unable to feed himself. His vocabulary had been gradually reduced to "yes" and "no," and later he was unable to utter these words.

April, 1897.—The muscular atrophy had become more marked. No improvement in his general condition had occurred. His limbs were contractured, the reflexes were much exaggerated, and the boy was unable to speak. Bed sores were present over both hips. He was unable to turn himself in bed.

Dr. Risley examined the boy's eyes, November 30, 1895, and found bilateral optic atrophy. It is not known how long this had existed.

Dr. White stated that the boy had a coarse tremor in voluntary movement. The impairment of gait was first noticed in the difficulty the boy experienced in going up and down stairs. His speech at first had been lisping, later became scanning, and finally was lost.

The preceding notes were furnished to me by Dr. Frank White from the case-books of the Pennsylvania Training

School for Feeble-Minded Children. I saw the boy only once; this was on May 11, 1897, at which time I made the following notes.

The reflexes all over the body were much exaggerated, distinct ankle clonus could not be obtained on account of the contractures, but a tap on the Achilles tendon of the right side threw the foot into a tremor, consisting of movements of flexion and extension. This was not obtained on the left side. A tap over either patellar tendon gave an exaggerated knee-jerk, although the leg was not moved to any great extent on account of contracture. The exaggeration was especially noticeable in the contraction of the thigh muscles. The reflexes in the upper limbs at the wrists and biceps tendons were exaggerated. The chin-jerk was also unusually prompt. The muscles of the extremities were much contractured, and the legs could be extended passively on the thighs only to a right angle. The right forearm could be extended passively so as to form an oblique angle with the arm. The left forearm could be fully extended passively. No voluntary movement could be detected. A fine tremor was produced by any passive movement of the right hand. Pain sense seemed to be preserved in all parts of the body, but cerebration was very slow, and no response in any form to questions could be obtained. The muscular wasting was extreme in all parts of the body, and the boy was scarcely more than skin and bones. He was entirely unable to stand, as his lower limbs were extremely flexed, and he had no power to extend them. The bowels were constipated and an enema was necessary to obtain any movement. The urine escaped involuntarily and constantly. He uttered no distinct words, but whined continually. Fibrillary tremor in the muscles when at rest was not observed, probably because the atrophy was too great.

The boy died ten days after this examination, and Dr. White was able to obtain permission to remove only the cord. As I was not on duty as pathologist to the institution at this time, I am indebted to my colleague, Dr. Joseph Sailer, for the pathological material.

Such is the clinical history of the case so far as it could be obtained. A boy, born at full term, in normal labour, in a family in which feeble mentality had been observed, presented nothing striking to his parents until he went to school. He had at first a fair amount of intelligence, and could read

and write; had a lisping speech, and no very marked disturbance of gait. As he grew older mental failure, spasticity, tremor on movement, scanning speech, contracture of the muscles of the limbs, gradual abolition of the power of voluntary movement, and bilateral optic atrophy were noted. Most of these symptoms, according to the history, developed in the last three or four years of life.

The process was evidently one causing degeneration of the pyramidal tracts, and probably of the anterior horns of the spinal cord, on account of the intense muscular atrophy. It was a process of long duration—if we may assume that the boy went to school when he was seven or eight years old—lasting seven or eight years, inasmuch as the patient was fifteen years old at the time of death. Indeed, it is very probable that the degeneration began before the signs were noticed by the boy's parents and teachers.

It was, therefore, with considerable interest that I awaited the results of my microscopical examination. No one can regret more than the writer the inability to obtain the brain from this patient, but the spinal cord affords a very interesting study.

The crossed pyramidal tracts are degenerated from the medulla oblongata throughout the cord into the lumbar region, and some sclerosis is seen in the area of at least one direct pyramidal tract. The sclerotic tissue in the lateral columns extends forward somewhat beyond the area of the crossed pyramidal tracts, and is symmetrical on the two sides of the cord. These areas are not entirely degenerated, but contain many normal fibres. While this degeneration is unquestionably present, it does not equal in degree that resulting from a destruction of motor fibres at a higher level. The motor cells in the anterior horns of the cervical cord are nearly, or fully, as numerous as in normal cords, and most of them appear to be normal. The motor cells of the lumbar cord seem to be somewhat deficient in number. As the spinal cord was unfortunately hardened in Müller's fluid, Nissl's method could not be employed.

The anterior roots are certainly not notably altered. The anterior horns do not appear to be as rich in fibres as in

normal spinal cords. The method of Marchi reveals some black dots in the sections, but not sufficient to render a diagnosis of recent degeneration probable.

These are, therefore, the lesions of amyotrophic lateral sclerosis, or more correctly, of lateral sclerosis, for the changes in the peripheral neurons are not marked, as they are in the central.

I cannot say that symmetrical gross lesions were not present in the motor regions of the brain, but the clinical history is much against such a view, and the degeneration of the pyramidal tracts equals in intensity that which I have found in amyotrophic lateral sclerosis, and is less, to my eye, than that known as secondary degeneration from cerebral lesions. This seems to me an important observation, although, of course, the age of the child must be taken into consideration. The boy was born in a family in which the mentality was feeble, and doubtless inherited a central nervous system which was not of the best. The gradual development of great spasticity and paralysis after he began to attend school, and the absence of these signs in a notable degree before this period, do not render the existence of bilateral hæmorrhage, softening or similar lesions probable. The clinical history and the pathological findings are much in favour of the diagnosis of amyotrophic lateral sclerosis, and yet this disease has been thought to be one of adult life.

Luce has reported a case which he says proves by histological investigation that tabes dorsalis and amyotrophic lateral sclerosis may occur in childhood,[1] and he further states that such a demonstration of the occurrence of amyotrophic lateral sclerosis in childhood has not been previously given.

The interesting cases reported by Werdnig[2] and Hoffmann,[3] though not typical clinically of amyotrophic lateral sclerosis, show that this disease may exist in childhood. Hoffmann, in his last paper on this subject reports a case which he says represents the disease very fully. I quote it,

[1] LUCE, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xii., No. 1.
[2] WERDNIG, *Archiv. für Psychiatric*, xxii., *idem* xxvi.
[3] HOFFMANN, *Deutsche Zeitschrift für Nervenheilkunde*, iii., *idem* x.

therefore, as representing Hoffmann's latest views on this subject.

A child born of healthy parents, in normal labour, appeared to be in good health until seven or eight months old. Flaccid paralysis began at that time to be apparent in the muscles of the buttocks and thighs, and gradually extended symmetrically to the back, neck, shoulder, upper arm, forearm and hand muscles, as well as to the muscles of the legs. Atrophy and loss of tendon reflexes, and secondary changes in the joints and vertebral column were noted, but no fibrillary tremors were seen, and sensation and the sphincters were intact. The mentality was good. The facial, lingual and pharyngeal muscles were normal. Death occurred from secondary pulmonary disease. In other cases reaction of degeneration had been noticed.

The peripheral motor neurons below the hypoglossal nerves were symmetrically degenerated, the cells of the anterior horns were much diseased, and sclerosis of the crossed and direct pyramidal tracts, and of the lateral ground bundles was noted, but the degeneration did not extend above the pyramidal decussation.

Twenty-two cases of this peculiar hereditary form of muscular atrophy, according to Hoffmann, are on record, and necropsies have been obtained in four of these. In all four the peripheral motor neurons were much diseased; in three the antero-lateral columns were affected, though in less degree, and in all the cases examined *post-mortem*, simple muscular atrophy was noticed.

The heredity, so far as known, the flaccid paralysis and loss of tendon reflexes were not present in my case, and the explanation for the existence of the flaccid paralysis and loss of reflexes in the other cases seems to lie in the greater degeneration of the peripheral neurons.

The case of T. H. M. resembles more the disease which Strümpell[1] has recently made prominent. He reports two cases of this malady which began in childhood and resembled multiple sclerosis, but no very marked lesions were found

[1] Strümpell, *Deutsche Zeitschrift für Nervenheilkunde*, vol. xii., No. 2.

at the necropsy. He refers to two similar cases reported by Westphal in 1883. These are the only instances known in literature. The disease has commenced at the thirtieth, eighteenth, twelfth, and ninth year of life. The cause is unknown, but does not seem to be hereditary or familial. The patients have usually been normal before the beginning of the malady. The motility of the limbs is especially affected (spasticity, paresis, ataxia, tremor), in this pseudo sclerosis. Complete and permanent paralysis is rare, or at least late in appearing, but transitory paresis is very characteristic, and in some cases apoplectiform attacks with temporary hemiplegia are observed. The tremor is prominent and occasionally is of a rhythmical character. Distinct nystagmus was not seen in any of the cases. Scanning, indistinct and explosive speech was observed. The facial muscles appeared in some cases peculiarly rigid. Epileptiform attacks have been observed. The muscular tonus is exaggerated especially in the legs, and the gait is paretic, ataxic, and spastic. Exaggeration of the tendon reflexes is characteristic. Sensation is little affected. Vertigo and disturbance of the vesical, rectal and sexual functions are not usually observed. Mental symptoms are never entirely absent, and forced laughter and crying are noted. The duration of the disease is long and remissions occur. It does not resemble hysteria.

The findings in Strümpell's two cases were negative, except that a very slight degeneration of the upper part of the lateral motor tracts of the cord was observed, but this was not sufficient to explain the symptoms. In two of the four cases the white matter of the brain was unusually firm.

Strümpell reports another case which he observed in 1879-1881, in a child of 11 years, when first seen, which resembles the pseudo-sclerosis. At the necropsy the consistency of the brain was greater than normal, and the crossed pyramidal tracts were slightly degenerated throughout the cord. It is possible that syphilis bears some relation to the disease.

Strümpell, therefore, observed slight degeneration of the lateral motor tracts of the cord. In my case this

degeneration was very marked, but the difference histologically between Strümpell's cases and my case seems to be largely one of degree.

The clinical history in my case pointed to a symmetrical progressive degeneration of the motor tracts, and the necropsy did not disappoint the expectations. If any cortical lesion was present it has escaped detection, and the report is imperfect to that extent. Such clinical cases are rare, and I feel justified in reporting this one, although the histological study was of necessity deficient.

The optic atrophy and the progressive mental enfeeblement bring to mind the affection first described by Warren Tay, and now known widely as amaurotic family idiocy of Sachs. Some of the other features are unlike the published cases of this disease.

It might be allowable to class this case among the cerebral diplegias, and there can be no doubt that the central nervous system was abnormal. Encephalitis might be thought of, but the clinical history does not seem to harmonize with such a diagnosis, unless we speak of the chronic form of this disease, which is not always easily distinguishable from primary degeneration of nerve cells and nerve fibres.

ON PARALYSIS AND MUSCULAR ATROPHY IN TABES DORSALIS, WITH OBSERVATIONS ON THE RELATION OF AFFERENT IMPULSES TO MOVEMENT AND MUSCULAR NUTRITION.

BY ARTHUR J. WHITING, M.D., M.R.C.P.

Registrar to the National Hospital for the Paralysed and Epileptic, Queen Square.

ALTHOUGH most of the prominent symptoms of locomotor ataxia are sensory, or at least afferent in their nature, as for example the anæsthesia and the lightning pains, there are yet some quite characteristic effects of the disease that are motor, or at least efferent, as for example the muscular palsies and atrophy concerning which this paper is written, and even, in the last analysis, the ataxy. In 1875 Hughlings Jackson[46] suggested that the ataxia of tabes is due to paralysis of certain highly special movements; but in 1896,[47] returning to the same subject, he writes, "I should have said paralysis, or rather, paresis *in effect*, the negative central lesion being a sensory one. There is in the early stage of the effort to walk (which effort, the operation being started by 'volition,' is from the highest cerebral centres) underdoing of the *movement* in which the peroneus longus *muscle* is chiefly engaged, and there is overdoing of the antagonist movement, that in which the tibialis anticus is chiefly engaged. I suppose the motor centre (lowest level) for the peroneal movement is not, consequent on the abrogation of the muscular sense nerves, 'informed' of the state of the muscles concerned in the movement, and is thus not kept 'ready to act,' and does not act efficiently in locomotion at the call of the

highest centres. There is 'paralysis in effect.' On strong efforts to make it act the associated centre for the tibialis anticus movement is made to overact."

According to Erb, Topinard, and Cyon,[2] distinct motor paresis can be demonstrated in at least one-half of all cases of tabes, while Duchenne found muscular atrophy in 8 per cent. of his cases, and Dejerine[33] in 19 per cent. of his. It was in 1832 that Cruveilhier[1] first described atrophy of the lower limbs and double club-foot associated with sclerosis of the posterior columns of the cord. Since then cases have been recorded in which there has been atrophy involving, in different patients, almost every muscle of the body. A more or less general atrophy, usually resembling the Aran-Duchenne type of progressive muscular atrophy, has been observed in tabetic cases by Virchow, Foucart and Duchenne, Friedreich, Charcot, Buzzard, Ballet, Eulenburg, Hacker, Fischer, Dejerine and Braun; atrophy of the upper limbs, and usually of the hands by Cuffer, Ballet, Condoleon, Remak, Hoffmann, and Dejerine; of the lower limbs, and chiefly of those below the knee, by Laborde, Dumenil, Leyden, Joffroy and Condoleon, Stransky, Goldscheider, and J. B. Charcot with Dufour; of the tongue by Charcot and Cuffer, by Ballet, Westphal, and Eisenlohr; of one of the laryngeal muscles by Dufour; of the facial muscles by Ballet; and of the muscles of mastication by Schultze, of Bonn. Cases of paralysis of muscles inaccessible to direct physical examination during life, such as those of the eye and larynx and the diaphragm, have been recorded by a still larger number of observers. In the majority of the cases with autopsies the atrophy has been associated with a neuritis almost entirely terminal; in one case recorded by Remak[24] there was neuritis of the right median nerve, in one by Goldscheider[36] neuritis of the sensory and motor nerves of the lower limbs, in another by Dejerine and Petreen, in which there was external ophthalmoplegia and paralysis of the larynx, there was a parenchymatous neuritis not involving the nerves between their nuclei and the ponto-bulbar surface. Dejerine[33] has recorded nineteen cases of muscular atrophy

occurring among ninety-three tabetics; in nine of these an autopsy was obtained, eight showing the Aran-Duchenne type of atrophy exhibited a well-marked neuritis principally of the intramuscular nerves of the extremities; the nerve changes were much less in the nerve trunks, and if present were only very slight in the anterior roots. In the ninth case, which was of the scapulo-humeral type, in addition to a neuritis there was, he thinks, a slight diminution in the number of anterior horn-cells in the cervical enlargement. In a minority of the cases with autopsies, the atrophy has been found to be associated with nuclear changes. Charcot[7] and Pierret[8] first directed attention to muscular atrophy in tabes accompanied with degeneration of the anterior horn-cells. There was in their well-known case atrophy of the muscles of the right upper limb, and more particularly those of the thenar and hypothenar groups, also of the muscles of the right side of the trunk and of the right lower limb. In this case the nerves themselves were healthy, and while the cells of the left anterior horn were quite normal, those of the right side, and especially the postero-external group, were greatly diminished in number, and those existing showed marked degenerative changes. They were able to trace degenerated fibres from the posterior roots to the anterior horns along the course of the so-called internal root-fibres, and they maintained that the cornual lesion had none of the characters of a polio-myelitis, but was in their opinion a degeneration secondary to that of the posterior columns.

In 1875 Cuffer[10] recorded a case of tabes in which during the prae-ataxic stage of the disease there had occurred atrophy of the right thenar muscles and right half of the tongue, with fibrillary twitching in the wasted tongue-muscles. An autopsy was made on this case in 1884 by Raymond and Artaud,[11] and it was found that in the dorsal and cervical regions of the cord the sclerosis had extended into the anterior horns, the cells of which were diminished in number, and those present shrunken, while in the bulb the right principal hypoglossal nucleus consisted of three or four cells alone as compared with thirty on the

left side, and these three or four showed degenerative changes.

In 1876 Jean[9] recorded the case of a tabetic patient in which there were laryngeal and gastric crises, with spasm of the pharyngeal muscles and muscular twitchings of the lower limbs coinciding with severe lightning pains. There was found *post-mortem* atrophy of the left vocal cord and of the left thyro-arytenoid muscle, with degeneration of the left vagal and spinal accessory nuclei, and atrophy of their roots.

In 1877 a case of tabes, with muscular atrophy associated with anterior horn changes, was published by Leyden.[45] The atrophy involved the limbs, but more especially the hands, and the serratus magnus, the greater pectoral and the dorsal scapular muscles on each side. There was also observed fibrillary twitching in the thenar and tongue muscles. The anterior roots proved to be healthy; the anterior horn-cells of the cervical and lumbar regions were atrophied, and there was an intra-muscular neuritis.

In 1882 Buzzard[13] recorded a case of ophthalmoplegia externa occurring in a tabetic patient, and Bevan Lewis found a lesion of the sixth nucleus, which he believed to be thrombotic. In 1881 Kahler[12] found degeneration of the inferior vagal nucleus in a case of tabes, with paralysis of the muscles of the pharynx and right vocal cord, and with gastric crises. Landouzy and Dejerine[15] (1883) discovered atrophy of the vagus-accessorius nuclei in a case of tabes with intense laryngeal crises.

Eisenlohr[16] (1884) has described atrophy of the hypoglossal nuclei, the vagus-accessorius nuclei and the right sixth nucleus associated in a case of tabes with fibrillary twitchings and atrophy of the tongue, paralysis of the palate, lips, laryngeal muscles, and right external rectus muscle.

P. D. Koch, of Copenhagen, and Pierre Marie,[18] examined in 1888 the medulla from one of three cases of hemiatrophy of the tongue in tabes that had been recorded by Ballet[17] in 1881. In addition, there was in this case

atrophy of the deltoids, of the muscles of the limbs (particularly the intrinsic muscles of the hands and those below the right knee), likewise paralysis of the soft palate and of the vocal cord on the same side as the wasting of the tongue. There was found atrophy of the right hypoglossal and spinal accessory nuclei. Westphal[27] published in 1887 a case of locomotor ataxia in which there was external ophthalmoplegia and atrophy of the left half of the tongue with weakness of the palate. Microscopic examination revealed degeneration of the third, fourth and sixth nuclei on each side, and of the left hypoglossal nucleus. In the same year Condoléon[26] made an elaborate study of a case of tabes with amyotrophy which had been previously recorded by Joffroy.[27] There was double club-foot; the muscles below both knees were wasted, particularly the peronei and solei, and more on the right side than the left; and there was atrophy of the intrinsic muscles of the left hand, more especially of the thenar and first interosseous muscles. In this case also severe lightning pains were often accompanied by involuntary contractions of the muscles, mostly of the hamstring group. Fibrillary twitchings were not observed. The electrical excitability of the wasted muscles was greatly reduced, but there was no characteristic reaction of degeneration. Degenerative changes were found in the anterior horn-cells of the eighth cervical and first dorsal segments of the left side, and their number was reduced, as compared with the right side, in the ratio of seven to ten. In the lumbar enlargement the anterior horn-cells were likewise decreased in number and degenerated, more, it was noted, on the right side than the left, corresponding with the muscular atrophy. The peripheral nerves were greatly degenerated, but the larger nerve-trunks and the anterior roots showed but slight changes. The degenerated posterior root-fibres could be traced as far as the anterior horns but not actually into their grey matter. A case in many respects similar was published by Braun[28] in 1888; there was in it atrophy of the intrinsic muscles of the right hand, the extensors of the right wrist and fingers, of both greater pectorals,

the supra-spinati, infra-spinati and deltoids. The wasted muscles showed the reaction of degeneration, and fibrillary contractions were seen in those of the forearm. There was found *post-mortem* numerical diminution and degenerative changes in the existing anterior horn-cells of the lower cervical region with degeneration of the lower cervical roots on the right side, also of the right radial, left sciatic, and right peroneal nerves.

In 1896, Schaffer[41] examined with the aid of more recent methods the spinal cord from a case of tabes with club-foot and atrophy of the peroneal muscles. While the anterior horn of the cervical cord was normal in the lumbar region, Nissl's stain revealed chromatolytic changes in the cells, a finely granular chromatin with vacuolation, giving an appearance, he states, similar to that produced in the nerve cells by poisoning with phosphorus or lead. The anterior nerve-roots were found to be perfectly healthy.

The two following cases illustrating the occurrence of muscular atrophy in locomotor ataxia are those of two of Dr. Ferrier's out-patients, who kindly allows me to record them in this paper.

Case I.—B. C., aged 44, began to suffer from "rheumatic pains" in the lower limbs some twelve years ago. The pains, which were "momentary," "shooting," or "shock-like" in character, recurred usually every three or four months, each attack lasting for three or four days at a time. Four years later (1890) he began to have difficulty in getting his left hand to the back of his neck, experienced chiefly in his daily ablutions, and about the same time he noticed difficulty in lifting things from the table. Five years ago (1893) he first came as an out-patient to the National Hospital, when Dr. Ferrier found that he had greatly contracted pupils which did not react to light, absent knee-jerks, and paralysis with wasting of the flexors of the left forearm. It was also found that the biceps and supinator longus did not react to faradism, although they gave KCC>ACC, while all the other muscles reacted normally. His disability slowly increased, and five months later (December 6, 1893) the left deltoid and infra-spinatus were found to have lost their faradic excitability and to give ACC>KCC. On May 16, 1894, the biceps still gave KCC>ACC without any response to faradism.

About this time he began to have girdle sensation, as of a rope round his waist, and to have difficulty in passing water. On February 18, 1896, the infra-spinatus, deltoid, biceps, brachialis anticus, and supinator longus, of the left side, gave no reaction to either faradism or galvanism, but the left supra-spinatus reacted well to faradism and gave KCC>ACC, and KCC with 5 m.a. of current. Latterly the shooting pains have been more severe and have involved his arms as well as his legs; but he has throughout been free from all crises and has not had any diplopia or staggering in walking even in the dark.

There is nothing of importance in his family history. At the age of 20 he had a chancre with well-marked secondary manifestations. He has not had any other illness with the exception of influenza five years ago. He has always been a temperate man and his work has been carried on indoors.

He is a well-nourished, muscular man, about 5 feet 1 inch in height and 9 stone 6 lbs. in weight. Although his complexion is pale, there is no evidence of general anæmia. His pulse rate is 104 per minute; it is small and slightly irregular, both in force and time. His rate of breathing is fifteen to the minute and is abdomino-costal in type. He is a quiet man, of good general conduct, without delusions or hallucinations, and is indeed of a considerable degree of intelligence. He can neither abduct nor advance the left humerus, nor flex the elbow joint, except very feebly by means of the long flexors of the wrist and fingers. When the elbow is extended the joint is locked with a jerk. Supination and pronation of the forearm are good. The left infra-spinatus, deltoid, biceps, brachialis anticus, and supinator longus are completely atrophied (see photographs 1, 2 and 3) and react neither to faradism nor galvanism. The trapezius, the rhomboids, the supra-spinatus, the pectoralis major, the serratus magnus, the triceps and the muscles of the left forearm, other than the supinator longus, act well, as also does the diaphragm. The right grasp is 110 (average normal about 155), and the left 35 (average normal about 145). The other muscles tested give normal reactions. His gait is probably unaffected; he may possibly bring his right heel rather more heavily down on the ground than the average person. He can "toe and heel" a line quite well. He stands steadily with his feet close together; if he shuts his eyes he sways slightly, but little, if any, more than the average healthy person. Co-ordination of movement generally is excellent. The unatrophied muscles are extremely irritable to direct percussion.

FIG. 1.

B. C.—Showing atrophy of the left deltoid, biceps, and supinator longus.

FIG. 2.

B. C.—Showing atrophy of the left deltoid, biceps, and supinator longus, also less clearly of the left infra-spinatus.

FIG. 3.

B. C.—Showing atrophy of the left infra-spinatus and deltoid.

The lightning pains are localised to the front of the thighs, the knees and the upper limbs. The girdle sensation is not constant. There is no paræsthesia or numbness. The sensibilities to touch, pain, heat, and cold are perfectly acute all over the body. Forcible compression of the ulnar nerves at the elbows causes only slight tingling in the little fingers. Sense of position is good at all the joints. There are no obvious vasomotor or trophic changes other than the muscular atrophy, except very slight grating on passive movement of the left shoulder-joint. The superficial reflexes generally are very brisk. The knee jerks cannot be elicited; the wrist jerks are fair; the right biceps-jerk is good, the left is absent. Neither the triceps jerks nor the jaw jerk are present. Except for slight tardiness of micturition the sphincters are unaffected. Smell and taste are good on each side. Hearing is about $\frac{10}{40}$ with each ear; a tuning fork held in the middle line of the head is heard equally well on the two sides, and with each ear air conduction is better than bone conduction. Vision is good, L. $\frac{6}{6}$, R. $\frac{6}{8}$; the visual fields are not restricted; colour sensation is good with each eye; the optic discs are of a good pink colour. The ocular movements, including convergence, are good. The pupils are small and unequal; the right measures 2 mm. in diameter, and the left 2·5 mm.; they do not react to light; they contract readily on convergence; they do not dilate when a painful stimulus is applied to the skin. The muscles of mastication act well, and the sensibility and movement of the face are good. The palate moves well and equally on the two sides. The larynx is normal (Sir Felix Semon). The tongue is protruded slightly to the right; it is not atrophied. There is nothing abnormal in the cranium or spine; the body organs are healthy, and the urine is normal.

This paralysis of the upper arm type obviously is not due to any peripheral lesion, because the paralysed muscles are supplied by several different nerves, which nerves supply additional non-paralysed muscles; moreover, there is no anæsthesia, although there are lightning pains both in the arms and in the legs, indicating an irritative process affecting the sensory tract. Having regard to the distribution of the atrophy, it may be held to be due to a lesion either of the anterior nerve-roots or of a group of nerve cells in the anterior horn of the cervical spinal cord.

It is probably not an anterior root-paralysis because with complete atrophy of some of the muscles supplied by the fifth and sixth cervical roots there is little if any weakness of other muscles supplied by the same roots; and results of autopsies in cases of tabes with muscular atrophy agree in showing that the anterior roots are very slightly affected, if at all, when the peripheral motor nerves are greatly changed, and even when in the same case both they and the corresponding anterior horn-cells show profound alterations.

Case II.—The second case is that of a man, D. E., aged 38, who was quite well until April, 1895, when he began to have constant numbness of his right thumb and index finger. Six months later his right little finger drooped and he was unable to straighten it. Two weeks after he noticed that his right thumb was weak and that the muscles of the ball of the thumb were smaller than previously. About the end of the December of 1895 he commenced to suffer from shooting pains in both sides of the back of the head, and a few weeks later in the left arm. In March following (1896) he found difficulty in getting his left hand up to his head when he wished to comb his hair; he had "terrible shooting pains" in the left shoulder, and he noticed a falling away of the muscle of the shoulder, of the front of the upper arm, and to a slight extent of the outer side of the forearm. Six months later (October, 1896) his right hand dropped at the wrist and at the same time he began to have pains in the right knuckles and numbness affecting the whole hand. About this time, too, he noticed an area of numbness on the side of the chest in the region of the left lower axilla. He has of late occasionally had a sensation of "drawing together" around the waist. For nine months virile power has been absent; on three occasions he has had incontinence of urine, but he has never had any trouble with the rectal sphincter. There has never been any diplopia, or difficulty in walking, or crises, or joint changes. His previous health has been excellent, except that eighteen years ago he had an attack of gonorrhœa; there were no secondary symptoms. He has always been temperate. There is no history of gout or rheumatism and there has been no special contact with lead. There is nothing of importance in the family history.

He is a moderately well-nourished man with fairly bulky but

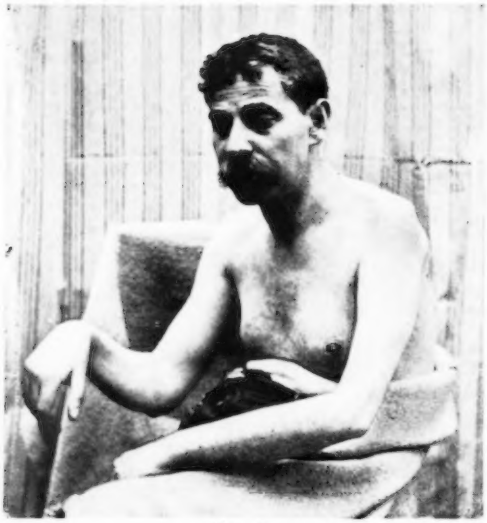


FIG. 4.

D. E.—Showing atrophy of the left deltoid, biceps, and supinator longus, also right wrist drop.

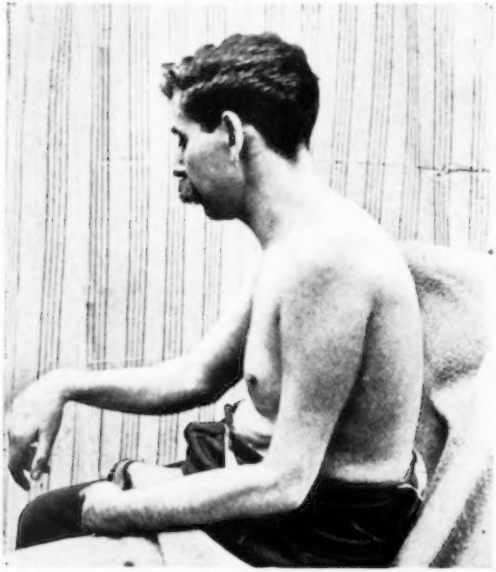


FIG. 5.

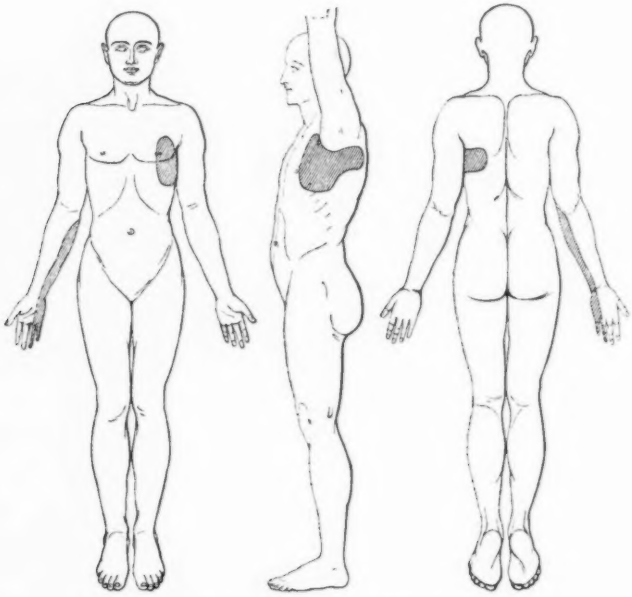
D. E.—Showing atrophy of the left deltoid, biceps, and supinator longus (and none of the infra-spinatus), also atrophy of the muscles on the extensor surface of the right forearm.

distinctly flabby muscles. His height is 5 ft. 5 in., and his weight 9 st. 7 lbs. His expression is anxious and depressed. There is no evidence of anæmia. His hair is streaked with grey. His pulse is regular but small and of low tension; its rate is 81 per minute. His mental state is excellent, except that he is inclined to be, perhaps, rather more despondent than circumstances warrant. His speech and articulation are unaffected. He is a left-handed man. There is right wrist-drop. All the muscles of the right thenar eminence are weak and they are all wasted, but more especially the adductores pollicis; he cannot touch the base of his little finger with the tip of his thumb. The hypothenar muscles are strong. The interossei act feebly. The long extensors of the fingers are paralysed and atrophied, but the extensor ossis metacarpi pollicis contracts feebly on volition. The extensors of the wrist are weak. The other muscles of the right upper limb act powerfully and are of good bulk. The intrinsic hand muscles give a reduced reaction to faradism, and the long extensors of the digits do not give any, while the extensor ossis metacarpi pollicis and the extensors of the wrist react well. To galvanism all these muscles give a reduced but brisk response with KCC>ACC.

He cannot abduct the left humerus and he flexes the elbow only very feebly, and that by means of the long flexors of the wrist and fingers alone. Pronation and supination of the forearm are good. When the elbow is extended the joint is locked with a jerk. There is complete atrophy of the deltoid, biceps, brachialis anticus, and supinator longus (see photographs 4 and 5), and these muscles do not react either to faradism or galvanism. The supra-spinatus, infra-spinatus, trapezius, the rhomboids, the pectoralis major, the serratus magnus, the triceps, the coracobrachialis and the diaphragm act quite strongly. The gait is not at all ataxic; and in spite of his muscular weakness he can dress himself and button his own clothing.

From time to time he has (1) "gnawing" or "drawing" pain in both arms, lasting for a few minutes; (2) stabbing, shooting pains in the shoulders, back, and upper limbs, and more on the right side than the left; (3) momentary drawing-pain in the back and in the right side of the chest in the region of the lower axilla; and (4) shooting pains in the lower limbs. There is subjective numbness of the whole of the right hand and of the lower axillary region of the left side of the chest. The ground feels natural to his feet. He occasionally has girdle sensation about the level of the navel. There is anæsthesia to

light touches along the ulnar border of the right forearm and hand, affecting the whole of the little finger and the ulnar half of the ring finger. There is also over this region (shown in the diagram) blunting of sensibility to painful and thermal stimuli. A similar loss of sensibility exists over an area (shown in the diagram) in the left lower axilla, extending to the mammary line in front and nearly to the angle of the scapula behind. While



D. E.—Loss of sensibility to touch, pain, heat and cold.

the left ulnar nerve is sensitive to pressure applied at the elbow the right is not. Sense of position is good at all joints.

The plantar reflexes are brisk, the conjunctival a little sluggish, and the pharyngeal very brisk. The knee-jerks are absent, as also are the elbow and wrist jerks. The organic reflexes are unaffected. There are no crises or joint changes or other trophic or vaso-motor alterations. The special senses are quite acute. There is no colour blindness. The optic discs

have a definite outline and a pink colour. The ocular movements are good, except convergence which is not well maintained with the right eye. There is no diplopia or ptosis. The pupils are unequal (right 6 mm., left 4 mm.), but they are regular in outline. They do not react to light, only feebly on accommodation, and they do not dilate on painful stimulation of the skin. The cranial nerves are otherwise healthy. The tongue is not atrophied. The cranium and spine show no abnormality. The body organs are healthy.

With the exception of the infra-spinatus the same upper-arm group of muscles are atrophied as in the former patient, but in this case the weakness in the first instance was associated with "terrible shooting pains in the left shoulder." Unlike the former case, there is loss of sensibility in the neighbourhood of the atrophy, but while the atrophy corresponds with the fifth cervical segment, the loss of sensibility corresponds with the fifth dorsal segment of the cord. Although one may, I think, without hesitation refer the upper-arm atrophy to a nuclear lesion, yet it is by no means so easy to form an opinion as to the origin of the atrophy in the forearm and hand of the other side. It does not correspond with an atrophy following a lesion of the ulnar nerve, inasmuch as all the thenar muscles are wasted (although the adductors of the thumb are more wasted than the rest, while the hypothenar muscles are not weak, neither is the inner half of the flexor profundus digitorum. While there is anæsthesia of ulnar distribution associated with weakness of some of the muscles supplied by the ulnar nerve, yet with atrophy of intrinsic hand muscles supplied by the median nerve there is no corresponding anæsthesia. The preservation of the extensor ossis metacarpi pollicis, while the other muscles supplied by the posterior interosseous branch of the muscular spiral nerve are atrophied (an escape not uncommon in lead paralysis), suggests a nuclear lesion. It is, therefore not improbable that the forearm and hand atrophy is not peripheral.

By the kindness of the physicians to the National Hospital for the Paralysed and Epileptic, I have been able to tabulate what I consider the important facts in relation

to the subject of this paper of 200 consecutive cases of locomotor ataxia that have been under their care in the wards of that hospital during the last twelve years. In 41 per cent. there was some definite paralysis of one or more of the external ocular muscles; in 12 per cent. there was diplopia but no other evidence of paralysis; in 5·5 per cent. there were nystagmoid jerkings without obvious paralysis; in many cases with definite paralysis there was nystagmoid jerking in addition; for example, in a case where all the ocular movements were bad except the outward movement, that was accompanied by nystagmoid jerking. In 85 per cent. the movements of the iris were abnormal, usually exhibiting inactivity to light alone, but sometimes inaction both to light and accommodation.

In 28 per cent. of the cases in which the larynx was examined with the laryngoscope there was some degree of paralysis of one or both vocal cords; in 20 per cent. of these there was in addition paralysis, or in one case merely fibrillary tremor, of half the tongue on the side corresponding with the laryngeal paralysis, and also in about a half (12 per cent.) of the latter cases there was observed paralysis of the palate on the same side.

In 4·5 per cent. of all the cases in which the larynx was examined there were laryngeal crises without any obvious paralysis; in 3·5 per cent. there were ataxic movements of the cords without paralysis; and in one case there were laryngeal crises with ataxia of the cords but no definite paralysis. The laryngoscopic examination in nearly all of these cases was made by Sir Felix Semon.

In 80·5 per cent. of the total number of cases there was vesical incontinence, or laboured micturition, or both; thus in 61·5 per cent. there was incontinence of urine, and in several of these cases it was noticed that sensation during micturition was good; in 19 per cent. there was either tardiness in starting the act, intermittent or feeble stream, or the patients complained of diffculty or strain in micturition, or of a lack of expelling power, some stating that they felt after each act that they had not completely emptied the bladder.

In 34 per cent. of all the cases there was defect associated with defæcation; in rather more than half it was of the nature of extremely obstinate constipation, in rather less than half there was definite rectal incontinence, in some cases only after the use of aperient medicines; while in several of the cases there was laxity, or gaping of the sphincter, or prolapse of the rectum, or other objective evidence of muscular insufficiency. In a few of the cases with and without gastric crises there was evidence of dilatation of the stomach, and at the autopsy in one case with crises it was found to be very greatly dilated.

In 30·5 per cent. of the cases there was demonstrable motor weakness of the skeletal muscles, usually of one or other limb, but this includes 4·5 per cent. of cases of hemiplegia. The principal facts of the more important of these cases are tabulated below. In this statement all cases with weakness that could not be verified objectively while in the hospital are excluded. In many cases excluded there was a history of transitory paralysis; and in many a complaint was made of sensations of heaviness during muscular exertion, of inability to walk far, of being easily tired, and occasionally of sudden loss of power in the legs. In a considerable number of the cases, although motor power was noted to be good yet the movements of the limbs were jerky. In about 6 per cent. of all the cases it was ascertained that the patients suffered from sudden involuntary movements during severe lightning pains. These usually affected the legs, but sometimes the arms, and in one case there were spasmodic contractions of the abdominal muscles in association with marked girdle pain.

Thus in 200 consecutive cases of tabes there are sixteen with obvious atrophy, or 8 per cent., the precise percentage given by Duchenne. It is probable, however, that in several of the cases without obvious atrophy the paralysed muscles were wasted. In the table of cases with paralysis but no visible atrophy, the only paralysis which is, as it seems to me, certainly not nuclear, is that of the right side of the face with loss of taste (Case 1), and in that case the paralysis of the vocal cords with the

No.	Name.	Age.	Duration.	Weakness or Paralysis.	Atrophy.	Electrical Reactions.
1	W. S.	34	6 yrs.	Left orbicularis palpebrarum; left external and superior recti; both masseters, temporals and pterygoids; left orbibularis oris; left half of tongue; soft palate; abductors of vocal cords; all muscles of neck; transitory of legs (for half-an-hour, with onset)	(Fibrillary twitching of orbicularis oculi on both sides); masseters and temporals of both sides; muscles of neck generally.	Orbiculares oculi react feebly to faradism and give A C C > K C C; no reaction in muscles of mastication; R. D. in left orbicularis oris; no reaction to faradism from soft palate; splenius capitis of both sides reacts feebly to faradism, the other neck muscles react well
2	C. J.	32	1½ yrs.	External eye muscles; orbiculares palpebrarum; orbiculares oris; all muscles of left face; right temporal and masseter	Almost complete of right temporal and masseter	Reduced response to faradism in muscles of left face; right orbicularis oculi; right orbicularis oris; right masseter and temporal
3	E. L.	39	2 yrs.	External ophthalmoplegia; abductors and internal tensors of vocal cords; right half of tongue (difficulty in swallowing); extensors and flexors of knees; abductors and adductors of thighs; diaphragm	Right half of tongue	Normal
4	B. C.	33	2 yrs.	Right external rectus; left half of tongue; abductors and internal tensors of vocal cords	Left half of tongue (anterior); deltoid and "arm muscles"	Normal
5	H. C.	37	2½ yrs.	Palate; left vocal cord; left trapezius; left sternomastoid	Left trapezius, with fibrillary twitchings	Left sternomatoid no reaction to faradism; L. trapezius feeble; to galvanism they give a sluggish response with A C C > K C C
6	E. W.	62	1 yr.	.. ..	Thenar muscles R. > L.; R. Extensors of wrist and fingers; R. Flexors of wrist and fingers; R. Triceps; R. Biceps, deltoid (posterior part); R. Supra spinatus, infra-spinatus; R. Thigh muscles; muscles below both knees	Reduced in all but K C C > A C C; no response to faradism from R. quadriceps extensa; R. Hamstring muscles; muscles below R. knee
7	T. H. C.	59	3 yrs.	Almost complete paralysis of both upper limbs; also both lower limbs, the hamstrings alone can be feebly contracted	Muscles of upper limbs, including deltoids and pectorals, but especially intrinsic muscles of hands; muscles of lower limb	Reduced response from wasted muscles but K C C > A C C
8	D. J.	52	12 mths.	Deltoids; flexors of forearms	Deltoids; scapular muscles	Reduced in the wasted muscles
9	M. E.	28	2 yrs.	Both hands, L. > R., especially of ulnar fingers	Intrinsic muscles of hands L. > R. (fibrillary twitchings in flexors of forearms)	.. ..
10	W. W.	52	12 yrs.	Bi-lateral paralysis of abductors and internal tensors of larynx; arms weak	Of muscles of forearms and hands, especially the interossei	.. ..

WITH MUSCULAR ATROPHY.

Ataxy.	Pain.	Sensibility.	Crises.	Trophic Changes.	Bladder weakness.	Rectal defect.
Ataxic gait; slight ataxy of hands	Shooting pains in legs	Blunted over face, soft palate, right side of the tongue, and in patches over the whole body, especially the lower limbs; muscular sense very defective in legs, less so in arms	Gastric; rectal; laryngeal	None	Tardy micturition; occasional incontinence	None
Ataxic gait; slight ataxy of arms	None	Patchy loss over face and limbs	None	None	Occasional retention and incontinence	None
Marked in upper and lower limbs	Shooting pains in legs	Patchy loss below second ribs and of ulnar borders of forearms and hands	Rectal; laryngeal	None	Tardy micturition; occasional incontinence	Occasional incontinence
Of upper and lower limbs	Shooting pains in legs and arms	Slight loss in legs and trunk	None	None	Incontinence	Incontinence
Marked of legs; slight of arms; Romhergism	Shooting pains in left leg	Patchy loss below second rib (especially on left lower limb) and on ulnar side of upper limbs; muscular sense lost from toes and left ankle	None	None	Tardy micturition	None
?None	None	Loss over right arm and both legs	None	Affection of right shoulder joint	Occasional incontinence	None
.. ..	Shooting pains in legs	No loss, but paresthesiæ of hands and arms—"pins and needles," "rawness"	None	None	Difficult micturition	Obstinate Constipation
Ataxic gait; Rombergism	Shooting pains in legs	Normal	None	None	Difficult micturition	None
Marked of arms; slight of legs	"Rheumatic" pains in R. knee and leg	Loss along ulnar borders of arms and across the chest	None	None	Occasional incontinence	None
Of legs and of hands	Shooting in legs	Slight delay of tactile sensibility; slight loss of muscular sense in right fingers	Gastric; laryngeal	None	Incontinence	Incontinence; prolapse of rectum

No.	Name.	Age.	Duration.	Weakness or Paralysis.	Atrophy.	Electrical Reactions.
11	D. B.	35	22 mths.	[Painless cramp in ulnar muscles of flexor surface of left forearm and of muscles of both legs; spontaneous movements of fingers]	Slight of left forearm muscles; of calf muscles with fibrillation	.. ..
12	K. E.	44	6 yrs.	Ophthalmoplegia; all movements of lower limbs weak but especially flexion of hips	Of L. thenar muscles and of the interossei of L. hand; also (?) of muscles below knees	Left thenar muscles react very badly to faradism; muscles below knees give slight diminution
13	L. T.	40	11 yrs.	Slight left ptosis; weak abduction of thumbs; dilatation of stomach (contents clear)	Lateral part of thenar muscles wasted	.. ..
14	S. G. M.	46	10 yrs.	Weakness of right side of tongue; weakness of all movements of lower limbs	Extreme in all muscles of lower limbs	Reduced faradic excitability in all muscles of lower limbs
15	B. T. W.	52	2 yrs.	Double ptosis; weakness of both legs L. > R.; paralysis of dorsiflexors of left ankle	Of all muscles of legs	.. ..
16	W. W.	37	2½ yrs.	Double ptosis; sudden paralysis of right thigh and leg; right foot drop	Of right anterior tibial and peronei, also in less degree the other muscles of right lower limb	R.D. in the right peronei, anterior tibial muscles and hamstrings

right half of the tongue and the transitory paralysis of the hand are quite probably nuclear. The paralysis of the palate, tongue and vocal cords (in Case 4) is almost certainly nuclear. In the remaining cases the origin of the atrophy is, I think, uncertain. In the table of "cases with atrophy" the weakness of the orbicularis palpebrarum (in Case 1) associated with paralysis of some of the external eye-muscles, and of the left half of the orbicularis oris associated with paralysis of the left half of the tongue, are almost certainly nuclear; also the ophthalmoplegia with weakness of the orbiculares palpebrarum in Case 2; and the atrophy of the deltoids and scapular muscles with paralysis of the flexors of the elbow joints. It is probably not possible to form a confident opinion as to the nature of the atrophy in the remaining cases, but it is likewise probably not possible to exclude a nuclear origin in any.

Ataxy.	Pain.	Sensibility.	Crises.	Trophic Changes.	Bladder weakness.	Rectal defect.
Jerky movements of legs	Shooting in legs; pain in legs with defecation	Loss over ulnar border of left forearm; hyperæsthesia of back; muscular sense in limbs impaired	[Spasmodic cough and dyspnœa following tingling over larynx]	None	Incontinence	None
None	Shooting in legs	Blunting with loss of muscular sense in both lower limbs	None	None	Tardy micturition	Obstinate constipation
Marked of legs; slight of arms	.. ..	Tactile anæsthesia up to level of third rib	?	None	Incontinence	Obstinate constipation
Marked in arms (cannot stand alone, if supported); gait very ataxic	Shooting in legs and arms	Lost over lower limbs and trunk; impaired in hands; muscular sense in legs lost	Gastric	None	Incontinence	Incontinence
Gait wildly ataxic	Shooting in legs	Anæsthesia of feet and perinæum; analgesia of lower limbs	None	None	Micturition difficult	None
Gait ataxic; flourishes right foot	None	Patchy anæsthesia of right leg; analgesia below level of second rib and along ulnar borders of arms	Gastric	None	Incontinence	None

THE INFLUENCE OF AFFERENT IMPULSES ON MOTOR MECHANISMS.

The well-known experiment of the "brainless frog" proves that after destruction of the cerebral centres there still remain some motor mechanisms in the spinal cord that may be excited to action by strong afferent stimuli, such as those produced by applying an acid fluid to the skin. These mechanisms represent combined movements, for the movements produced are quasi-purposive in character; and, moreover, their activity, it is to be presumed, is independent of nerves which habitually carry impulses that result in sensation. Both Sherrington[49] and Page May[50] have showed the remarkable fact that stimulation of the posterior roots (in the dog and monkey) causes contraction of groups of muscles. Page May found likewise that similar movements were produced by faradic stimuli

No.	Name.	Age.	Duration.	Paralysis.	Ataxy.	Pain.
1	B. J.	39	4 yrs.	Abductor paralysis of vocal cords; weakness of right half of tongue; paralysis of right face (with loss of taste); transitory paralysis of left hand; weakness of legs, sudden in onset	Gait very ataxic; hands slightly ataxic; Rombergism	Shooting pains in lower limbs
2	D. W.	43	4 yrs.	Paralysis of left vocal cord; paralysis of right masseter and pterygoids; paralysis of muscles supplied by both third nerves	Slight of arms and legs; Rombergism	Shooting pains in lower limbs
3	T. J.	57	1 yr.	Abductor paralysis of right vocal cord; double ptosis and weakness of all ocular muscles except inferior oblique which overacts	Marked in all limbs; Rombergism	Shooting pains in legs
4	C. J.	39	1½ yrs	Weakness of all muscles supplied by both third nerves; abductor paralysis of vocal cords; weakness of right half of soft palate; weakness of right half of tongue; weakness of right side of face	Gait very ataxic	Shooting pains in legs
5	T. W.	44	13 mths.	Cannot move feet at ankles; moves toes feebly; movement at knee joints feeble; weakness of flexors of left wrist and fingers	Slight of legs and arms	None
6	G. R.	36	3 yrs.	Weakness of lower limbs, especially of left ankle; clawed feet (spontaneous athetoid movements of fingers of right hand)	Gait ataxic; also right arm; Rombergism	Shooting pains in legs
7	B. H.	41	3½ yrs.	Weakness of all movements of lower limbs, especially flexors of hips (legs suddenly "gave way" has not walked or stood since)	Movements of lower limbs unsteady	Shooting pains in legs and simultaneously in lower abdomen, causing legs to draw up
8	R. E.	41	5 yrs.	Paralysis of dorsiflexors and evertors of left foot	Marked of legs; slight of arms	Generally distributed shooting pains
9	G. F.	42	20 mths.	Weakness of left anterior tibial group of muscles (faradic excitability reduced)	Gait slightly ataxic	Shooting pains in legs
10	H. M.	45	4 yrs.	Paralysis of dorsiflexors and evertors of left foot; weakness of left lower limb [R.D. in left tibialis anticus, ext. com. dig., ext. long hall. Reduced faradic excitability in the peroneï.]	Ataxy of leg; Rombergism	Shooting pains in legs, in trunk, and occasionally in arms
11	L. C.	53	5 yrs.	Weakness of dorsiflexors and evertors of right foot, also weakness of flexors and extensors of toes; weakness of evertors of left foot [right club foot]. (Faradic excitability much reduced in peroneus longus)	Ataxy of legs and hands	Shooting pains in legs with spasmodic flexion of knees; (movements of legs and right arm during sleep)
12	P. S.	54	6 mths.	Flaccid paralysis of both legs; double foot drop [no R.D.]; weakness of left external rectus	None	Shooting pains in legs; aching in coccygeal region
13	C. S.	44	6 yrs.	Weakness of dorsiflexors of left ankle	Of legs and arms; Rombergism	Shooting pains in legs

Dorsalis with Paralysis.

Sensibility.	Crises.	Trophic Changes.	Bladder.	Rectum.
Patchy loss below second rib; muscular sense impaired in lower limbs	Gastric; laryngeal	Charcot's joint; changes in both knees	Tardy micturition; incontinence	None
Loss over face	None	None	Tardy micturition; incontinence	None
Blunting in hands, feet and ulnar side of forearms	None	Perforating ulcer of foot	Tardy micturition	None
Patchy loss below second rib; loss over ulnar side of upper limbs; muscular sense lost in legs and little fingers	Gastric; laryngeal	None	Occasional retention and incontinence	None
Blunting in hands; muscular sense lost in legs and hands	None	None	Difficult	Incontinence (after aperients)
Blunting over lower limbs and right hand	None	None	None	None
Slight anæsthesia of legs	None	None	Retention; precipitate micturition	None
None	None	None	None	None
Blunting below knees	Gastric; laryngeal	None	Occasional incontinence	Occasional incontinence
Blunting below left knee; muscular sense defective in left great toe	None	None	Occasional incontinence	None
Loss over forearms, hands, and below middle of thighs	None	None	Occasional incontinence	None
Of lower limbs; of root distribution	Rectal	None	Incontinence	Tenesmus; gaping of sphincter ani; no anal reflex
Relative analgesia in arms	Gastric	None	Difficulty in micturition	None

applied to the surface of the cord in the region of the postero-external column (but not in the region of the postero-internal), and he showed that, although both flexor and extensor muscles were thrown into contraction, the "resultant" movement was one of flexion. On stimulating the postero-external columns of the lower lumbar segments he noticed rumbling of the bowels with closure and protrusion of the anus. It is possible that the spasmodic flexion of the lower limbs occurring in association with severe lightning pains may be analogous to the movements experimentally produced. Not only, however, does stimulation of the posterior roots result in movement, but conversely destructive lesions of the posterior roots produce paralysis. Claude Bernard[53] divided the posterior roots in frogs and dogs and demonstrated an ensuing paralysis of the corresponding limbs. The same phenomena of paralysis have been shown by Sherrington and Mott[48] to obtain in the monkey, and they proved that the paralysis depended more on the interruption of impulses coming from the skin (and particularly that of the palm and sole) than of those coming from the muscles. Tissot and Contejean[54] have made similar experiments with similar results; in one after section of the four lower lumbar and two upper sacral roots on one side there was not only complete paralysis of the posterior limb on that side, with absolute muscular relaxation, but also persistent incontinence of urine. In another experiment, after extirpation of the inter-vertebral ganglia of the same posterior roots, there was complete paralysis of the limb for four or five days, then the animal began again to use it, but with marked ataxy and pronounced overaction of the extensor muscles. In explanation of these results Sherrington and Mott suggest that the interruption of sensory impulses passing to the cortex cells is the cause of the paralysis; but, as Bastian[52] states, in those comparatively rare cases where the upward prolongations of the posterior root neurons are destroyed in the hinder part of the internal capsule, there is no paralysis, and he suggests that, as in lesions of the commissure between the auditory word-centre and the motor speech-centre, there

results an aphasia as absolute as that produced by lesions of Broca's convolution itself; so, after interrupting the afferent impulses that normally go by the reflex collaterals to the anterior horn-cells, there ensues a molecular change in these cells which blocks the passage of "voluntary" impulses from the cortex to the muscles. It is not difficult to find clinical phenomena that may correspond in their mechanism with the changes artificially produced; for example, the giving way of the legs in association with severe lightning pains, which, as Buzzard and others have stated, is truly a paralytic effect, the weakness of the sphincters, the inability to empty the bladder, the obstinate constipation, and the laxity of the sphincter ani.

That the muscle tonus is abolished by section of the posterior roots is a fact established by the experiments quoted, and as is well known (particularly through the observations of Fraenkel[55]), in tabes the tonus of the muscle is so much diminished that the limbs of the patient may be moved passively, without pain, into attitudes impossible in health; although at the same time it should be remembered the mechanical irritability of the muscle is greatly in excess of the average normal.

In addition to the loss of tonus, Mott and Sherrington[48] observed "a certain degree of wasting" of the paralysed muscles but "no appreciable alteration of colour"; Tissot and Contejean likewise found the muscles slightly atrophied. From the phenomena of the so-called arthritic atrophy one may infer that an irritative lesion of afferent nerves is more effective in producing atrophy than a paralysing one, for Raymond[56] showed that after the injection of an irritant, such as a solution of a mercury salt, into the knee joints, atrophy of the surrounding muscles ensued unless the posterior roots were divided. Massary,[56] following the suggestion of Dejerine, believes that the muscular atrophy of tabes corresponds essentially with that seen in hemiplegia; that for the exercise of the normal trophic energy of an anterior horn-cell on its neuron it is necessary that afferent impulses should reach it, on the one hand, from the cortex by a pyramidal fibre, and, on the other, from the peripheral

nerve ending in the skin, by the posterior root and reflex collateral; that when either channel is interrupted the nutrition of the cell suffers and muscular atrophy results. It is quite true that in some cases of hemiplegia there is marked atrophy, but one can hardly admit it to be comparable with that seen in tabes, chiefly because there is no loss of muscular tonus, or any approach to the reaction of degeneration, while the atrophy is never absolute so as to abolish galvanic excitability as may occur in tabes.

Observations on the changes that result in the anterior horn-cells after division of the posterior roots have recently been made by Warrington,[51] who found distinct evidence of chromatolysis of the cells, mainly of those of the postero-external group in the lower cervical and lower lumbar regions.

Schaffer,[44] as previously remarked, found similar changes after using Nissl's method in a case of tabes with atrophy of the peroneal muscles. Charcot[7] long ago traced, in a case of tabes with club-foot, marked degenerative changes from the posterior roots along the reflex collaterals to the anterior horns. Condoleon[26] particularly emphasised the fact that profound changes may exist in the anterior horn-cells together with marked degeneration of the intra-muscular motor nerves, and yet the larger nerve-trunks and the anterior nerve-roots be almost unchanged. As Schaffer points out, the failure of Dejerine to find cell degeneration in the presence of peripheral lesions loses its value as evidence because the observations were made before the more recent methods of demonstrating slight structural alterations in nerve cells were invented. It follows, then, that in some of the cases of muscular atrophy in tabes the lesion is nuclear, and in others it is not at present possible to exclude either a central or a peripheral lesion; and Dr. Hughlings Jackson's views, to which reference was made at the beginning of this paper, are confirmed by the discovery of chromatolysis of the anterior horn-cells. It would seem, therefore, that tabes is as much a segmental disease as a system disease, that it is indeed strictly a neuron disease.

I desire in conclusion to express my gratitude to the physicians of the National Hospital for the Paralysed and Epileptic for the generous manner in which they have permitted me to make use of the notes of their cases, and more especially to Dr. Ferrier for his kindness in allowing me to publish the two cases here recorded.

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ON TWO CASES OF EARLY SYPHILITIC PARAPLEGIA.

(A year after primary syphilis).

BY F. PARKES WEBER, M.D., F.R.C.P.

With Histological Report by J. S. Risien Russell, M.D., F.R.C.P.

So considerable a proportion of cases of acute spinal paraplegia give a history of primary syphilis about a year previously to the onset of the spinal disease, that one is forced to consider the paraplegia as really due to the syphilis, though *post-mortem* examination of the cord in such cases may show nothing which is absolutely peculiar to syphilis.

The following two cases deserve to be recorded, chiefly on account of the histological report kindly furnished by Dr. Risien Russell, to whom I am much indebted.

Case I.—Charles S., aged 23, was admitted to the German Hospital in June, 1896, suffering from paraplegia, bedsores, and hectic fever. Ten months ago he had a chancre, and four months later a rash appeared, the remains of which were still visible when he entered the hospital. For the six weeks previous to admission both his legs had been paralysed. He was unable to retain his urine and fæces. Sensation was absent up to about a hand's breadth above the umbilicus. Treatment by mercurial inunction and iodide of potassium did no good, and the patient died nineteen days after admission.

At the necropsy the spinal cord appeared natural or almost natural to the naked eye, and there was no obvious transverse softening to be found.

Dr. Risien Russell's Report.

In this case nearly the whole of the thoracic together with the lumbar and sacral regions of the spinal cord were available

for examination. Evidences of meningitis are seen both in the thoracic and lumbar regions, but the exact degree of this, and its extent, varies at different levels. In the upper thoracic region the meninges surrounding the whole circumference of the cord show marked signs of inflammation, while they are less uniformly involved in the lower thoracic region, and still less so in the lumbar cord. The meninges in these less affected parts show little or no change in some areas, while in others the change is pronounced, the distribution of the meningitis being somewhat patchy. The changes in the meninges consist in thickening with cell proliferation and exudation; the vessels are dilated, many of them engorged, and their walls thickened with multiplication of the nuclei.

The spinal cord itself is most pronouncedly altered in the upper thoracic region where marked myelitic changes exist; the nerve elements are greatly destroyed and are replaced by granular material, round cell proliferation and an increase of interstitial tissue. The vessels are much engorged, their walls markedly thickened, with increase of nuclei and perivascular proliferation of round cells. Other of the vessels are completely occluded, a fibrous cord without any evidence of a lumen being met with in such cases. Precisely similar changes are met with in the grey matter, where the round cell proliferation is even more pronounced than in the white, but not so great as in the immediate vicinity of the vessels in the latter. The shape of the grey matter as seen in mass is greatly altered, the distortion of it being most marked in connection with the anterior horns. Changes similar in kind, though much less extensive in distribution, as regards the amount of the transverse area of the cord affected, are seen in the lower thoracic region; and though the grey matter is involved as well as the white, the former preserves its characteristic shape instead of being distorted as in the upper thoracic region. Sections from the lumbar cord show still slighter changes, which can be regarded as the direct result of an inflammatory process. There is an increase of round cells in the grey matter and in the white matter of the lateral and ventral regions of the cord, and the interstitial tissue is thickened chiefly in the same parts. Many of the vessels have thickened walls, but few of them are engorged, only one or two here and there being found in this condition both in the grey and white matter.

The destructive changes of the nerve elements are, of course, most pronounced in the upper thoracic region, and here the

nerve fibres have suffered in greatest degree at the periphery of the cord and in the posterior columns, one half of the cord being damaged to a somewhat greater extent than the other. Sections prepared by the Weigert-Pal method show that in the most affected parts only scattered nerve fibres remain whose myelin sheaths are stained, and some of these are obviously swollen and are undergoing various degrees of degeneration, while the sheaths of other fibres take on the stain very imperfectly. Some of the swollen fibres are darkly stained throughout their whole transverse extent, there being no distinction between the axis-cylinder and myelin sheath. In addition to these changes, large blobs of variously altered myelin are to be seen, some partly stained and some quite unstained, while there also exist large and small empty spaces from which the products of degeneration have escaped, or have been removed. The white matter which bounds the grey is better preserved than is that at the periphery of the cord; but even here changes exist which are similar in character, though considerably less in degree. Some of the degenerated fibres in the posterior columns and antero-lateral regions of the cord are no doubt to be accounted for by ascending degeneration, the result of destructive inflammatory lesions in other segments nearer the caudal end of the cord, but the major part of the degeneration of the nerve elements can well be accounted for by the presence of the local inflammatory changes which have been already described.

The degenerative changes in the white matter diminish in amount as we pass toward the caudal end of the cord, so that about the mid-thoracic region the white matter of the periphery, though still more affected than that more centrally situated, is but moderately degenerated in some parts, especially in the ventral region of the cord. The lower thoracic region is very much better preserved, so that beyond some evidence of descending degeneration in the cross pyramidal tracts and in the antero-lateral region, only islets of degeneration of nerve fibres exist, the more important of which occupy the antero-lateral region, especially on one side, while smaller areas of the same kind exist in the posterior columns. With the exception of a few degenerate fibres here and there, the lumbar cord is free from degeneration of the nerve elements other than that which occupies the cross pyramidal tract regions and the anterior columns, and which is evidently the result of descending degeneration consequent on destructive processes in the thoracic cord.

The neurons of the anterior horns are profoundly altered, the

cells are completely destroyed in the upper thoracic region, where, with the exception of a very occasional much swollen and obviously degenerated cell, none are to be seen. The same may be said of the cells of Clarke's columns and of those of the posterior horns. Similar destructive changes in connection with the neurons exist in the caudal part of the thoracic cord, where, however, more of the cells have escaped total extinction, especially in Clarke's columns; but most of these are swollen and obviously degenerated, and only very exceptionally a fairly normal looking cell is seen. In marked contrast to the condition of the neurons in the thoracic region, those in the lumbar cord are well preserved; most of the cells appear quite normal, only one here and there showing any evidence of degenerative change, and that but slight as compared with what has already been described in connection with the thoracic cord.

Case II.[1]—R. W., aged 42, was admitted into the German Hospital in September, 1894. Patient had had a chancre and skin eruption a year before admission, in September of 1893. He had infected his wife, who was suffering from secondary symptoms at the time of patient's admission to the German Hospital. There was, therefore, no doubt as to the correctness of the history of syphilis. Six weeks before admission patient began to have difficulty in micturition, and some loss of power in lower extremities gradually showed itself. Three days before admission he suddenly became completely paralysed in the left leg. On admission there was complete paralysis of the left lower limb and motor paresis of the right one. Knee-jerks were absent. Sensation was very slightly if at all impaired. The bladder was completely paralysed, and had to be emptied at regular intervals. There was likewise incontinence of fæces. Two days after admission paralysis of the right lower limb became complete.

Treatment was begun on admission—mercurial inunction with $1\frac{1}{4}$ drachms of the unguentum hydrargyri daily, and iodide of potassium internally (15 grains thrice daily). After three weeks, when the patient had had eighteen inunctions, the treatment was left off, as it was found to have done no good. The paralysis remained stationary. Acute decubitis showed itself soon after admission, and this and cystitis were the ultimate cause of the

[1] This case was described in the Discussion, "On the Affections of the Nervous System occurring in the Early (secondary) Stages of Syphilis," at the Royal Medical and Chirurgical Society in 1895 (*Proceedings*, vol. vii., p. 108), but the cord had not yet been examined microscopically.

patient's death six weeks after admission. There was hectic fever during the last four weeks. Tactile sensation was not lost in the paralysed limbs, but special sensation for heat and cold was not examined.

At the *post-mortem* examination a transverse area[1] of softening was found in the spinal cord, which for about an inch was diffluent and pinkish in colour. The softened area with parts of the cord above and below it were removed for microscopic examination.

Dr. Risien Russell's Report.

Sections from the cervical and thoracic regions of the spinal cord were alone available for examination. These show evidences of meningitis; the meninges are thickened by exudation, the vessel walls are also increased in thickness with multiplication of nuclei, and many of the vessels are dilated, some being full, while others are empty, and some of the latter are flattened so that opposite sides of the inner wall of the vessels are in contact or almost so. No occlusion of any of the meningeal vessels by a process of endarteritis is to be seen. The only sections in which the meninges are sufficiently well preserved for satisfactory study of these changes are those of the thoracic region below the chief seat of softening; but there is enough evidence from the other sections to make it certain that meningitic changes also exist in the upper regions of the cord.

The sections show that the spinal cord itself is greatly destroyed in one part; much of this is the result of myelitis, but a good deal is also due to the unavoidable damage which has been done to this softened part during the process of preparation for microscopic examination. It is not possible to differentiate grey from white matter, and the general characters of the sections are so altered that it is impossible to say from what region of the cord it has been taken; but an examination of sections from parts of the cord above and below the lesion makes it clear that this chief area of softening must have existed in some part of the upper or middle region of the thoracic cord. The nerve elements are profoundly altered in this part, the myelin sheaths are seen in various stages of degeneration, and nothing resembling a nerve cell can be seen. There is exten-

[1] I was not present at the *post-mortem* examination, and at the Society was doubtless wrong in stating that the chief area of softening was in the lower dorsal region. Dr. Russell's report shows that it was in the upper or middle thoracic region.

sive exudation, with marked vascular engorgement, and actual hæmorrhages in some places. In spite of these pronounced myelitic changes, however, there are many nerve fibres whose myelin sheaths have escaped destruction; in such the differentiation between myelin sheath and axis-cylinder is preserved, but even these fibres stain badly.

Though the part of the cord just described is that chiefly affected, the process is not simply a transverse myelitis affecting this region and it alone. The change is a much more widespread one, a similar state of things, slighter in degree, existing in the cervical cord, and in the thoracic region below the chief seat of softening. In the cervical region, which is more affected than the lower thoracic, the change is most marked in the lateral regions of the cord, and more so on one side than on the other, while the posterior columns are only slightly affected, and there is no evidence of ascending degeneration in these tracts. Most of the vessels are engorged, and as a rule their walls are thin; but there are no hæmorrhages. Here and there a small vessel is seen with thickened walls and its lumen much narrowed; but no actually occluded vessels are seen. The grey matter of the anterior and posterior horns show similar changes: exudation, engorged vessels and even hæmorrhagic extravasations. A few of the anterior horn cells are preserved, but a great many have been destroyed, especially on one side.

In the thoracic cord below the chief seat of softening there is evidence of descending degeneration in both crossed pyramidal tracts, and in the direct pyramidal tract of one side, the opposite direct tract showing little or no change. In addition to this there is some evidence of some inflammatory change, but this is very slight, and in no way to be compared with that met with in the upper regions of the cord.

The absence of ascending degeneration in the cervical region of the cord, and the presence of descending degeneration in the lower thoracic region, make it probable that the myelitic process did not affect the posterior columns to any extent at any level below the part of the cervical cord examined; but that the chief brunt of the mischief must have fallen on the lateral and ventral regions of the cord. Moreover, the absence of descending degeneration in one direct pyramidal tract makes it clear that one ventral half of the cord must have suffered in greater degree than the other at a period sufficiently remote for descending degeneration to result.

Remarks.

The vascular changes which have been claimed as typical of syphilitic myelitis were present to a marked degree in Case I. but are comparatively absent in Case II. This constitutes probably the most important histological difference between the two cases. In fact, in the first case the degeneration of the nerve fibres seems to be due largely to the cutting off of their blood-supply owing to the vascular affection, whilst in the second case it seems to be due chiefly to some direct action of the syphilitic poison on the nervous elements themselves. Cases of the first kind have been described by E. Lanceraux,[1] H. Lamy,[2] E. Biernacki,[3] and others. Although T. Rumpf[4] suggests that many cases of so-called acute ascending paralysis are really due to syphilitic vascular changes, even Lanceraux[5] acknowledges that syphilis is probably not the only disease which can give rise to obliterative changes in the spinal vessels, and in Biernacki's[6] first case there was no history of syphilis. On the other hand my second case shows that the vascular changes in the cord are not always a chief feature of syphilitic cases. In fact as Dejerine[7] maintains, syphilitic cases of acute spinal myelitis cannot yet be distinguished symptomatically, during life, or pathologically, after death, from non-syphilitic cases, except by the history of fairly recent syphilitic infection.

At one time it was sometimes maintained that there was no such thing as a diffuse syphilitic affection of the spinal cord without grave meningeal changes, but that all were cases of meningo-myelitis. Though undoubtedly many of the published cases are examples of this, other cases show

[1] *Vide Leçons de Clinique Médicale*, Paris, 1892, p. 217.
[2] "De la méningo-myélite syphilitique," in *Nouvelle Iconographie de la Salpêtrière*, 1893, vol. vi.
[3] "Myelopathia endoarteriitica acuta," in *Deut. Zeitschr. fur Nervenheilkunde*, 1897, vol. vii., p. 173.
[4] *Die Syphilitischen Erkrankungen des Nervensystems*, Wiesbaden, 1887, p. 340.
[5] *Loc. cit.*, p. 218.
[6] *Loc. cit.*
[7] "De la myélite aigüe centrale survenant chez les syphilitiques à une époque rapprochée du début de l'affection"; in *Revue de Médecine*, Paris, Jan., 1884, p. 60 and p. 76.

that the syphilitic spinal affection can exist without the meninges being macroscopically much affected. In such cases the parenchymatous affection of the spinal cord is not secondary to meningitis.[1] My first case at least may be cited as an example of this.

It is noteworthy that in the first case no distinct macroscopic lesion could be detected in the cord. A similar fact is noted in certain other recorded cases, as in those described by W. Möller[2] and H. Schmaus.[3]

The importance of early treatment in all similar cases need hardly be remarked on. Anti-syphilitic treatment in very acute cases, unless commenced early, may be powerless to avert a fatal result. In fact, in the very acute cases of diffuse spinal meningitis following syphilis (which usually appears early, often in the first year after primary syphilis), presenting the clinical features of a transverse myelitis, an altogether gloomy view of the prognosis is taken by some authors, even granting that anti-syphilitic treatment be carried out.[4] A. Boettiger[5] says that in these very acute cases death generally results in the course of a few weeks or months in consequence of such complications as pyelonephritis, bed-sores, erysipelas, &c. Gilles de la Tourette[6] has apparently a more favourable opinion of the results of treatment, provided that it be commenced energetically and without delay.

[1] Compare A. Boettiger "Beitrag zur Lehre von dem luetischen Rückenmarkskrankheiten," in *Archiv für Psychiatrie*, 1894, vol. xxvi., p. 690.

[2] See the abstract by G. Thibierge in *Annales de Derm. et Syph.*, 1891, vol. ii., p. 352.

[3] "Zur Kenntniss der Rückenmarkssyphilis," in *Deutsches Archiv für Klin. Med.*, 1889, vol. xliv., p. 246.

[4] Compare F. Scalfati, in *La Riforma Medica*, 1895, vol. i., p. 122.

[5] *Op. cit.*, p. 690.

[6] "Formes cliniques et traitement des myélites syphilitiques," in *Le Bulletin Médical*, Paris, June 10, 1896.

AN EXPERIMENTAL STUDY OF VISIONS.

BY MORTON PRINCE, M.D.

Physician for Diseases of the Nervous System, Boston City Hospital.

THE study of visions is of interest from many points of view; thus it would be interesting to understand the origin of the visions which have influenced the actions of certain historical characters, such as Joan of Arc and Catherine of Sienna. It is said that even Bismarck had a vision which he believed to be supernatural.

It ought to interest the theologian to understand the psychical origin of visions, like those of Luther, Savonarola, and hosts of saints, which have had such influence upon theological ideas. If it had been understood that such visions were only the pictorial representation in consciousness, according to natural psychical laws, of the fleeting thoughts, prayers, or beliefs perhaps long forgotten, with which the religious enthusiast had occupied his mind at one time or another, the influence of the Church might have been differently exerted.

For those interested in psychical research, a study of visions may tend to throw light on many seemingly "occult" phenomena; and for the psychologist, it may be possible to learn, from a study of the laws of the development of visions, other facts regarding the organisation of the mind; while, finally, by such a study, the alienist may be enabled to better understand the hallucinations of the insane. So that from many points of view the subject presents a field for interesting research.

As the nervous system acts by general laws, what is true of the origin and mode of development of vision is probably true of hallucinations of the other senses, of which those of

hearing are the most important; so what we have learnt true of one, we can predicate as likely to be true of the other. Thus, a study of visions may also enable us to understand those hallucinations of hearing, which, like visions, have figured so conspicuously in the religious history of the past, and which play so large a part in the diseases of the mind.

It is well known that some people have the faculty of being able to create visions at will by intently gazing at an object. A crystal ball is commonly used for the purpose; the subject gazes into the crystal and, after a few moments of mental concentration, sees something therein—a scene, a person, or anything else, as the case may be. The phenomena have been called crystal visions from their mode of production. They are not particularly rare, and some, though not very many, experimental observations have been reported. In former times these visions played a part in occult lore, and, like modern clairvoyance, served to impose on the credulous. So that this part of psychology had its prototype in mysticism, just as chemistry may trace back its origin to alchemy, and astronomy to the romances of astrology. It is not so well known that it is the fashion for "spiritualistic mediums" to artificially cultivate the art of seeing visions, the method in use for the purpose being practically that of crystal gazing.[1] The hallucinations of hearing, which these same mediums profess to be communications with spirits of another world (and which some otherwise intelligent people believe), probably have an origin similar to that of visions, and are worthy of study from the point of view of this hypothesis. Visions created artificially by crystal gazing probably do not differ in their mode of

[1] A patient of mine having the curiosity to learn something about mediums, undertook (against my advice) to have herself developed by a professional. The "professor" claimed to have developed a very celebrated medium who has since been the object of study by Dr. Hodgson, Professor William James, Professor Lodge, and others. My patient told me that her education consisted in gazing intently, in conjunction with the professor, at a spot in the middle of a table, until she saw a vision at that spot. This was repeated at each lesson. If I remember rightly, a low diet and fatigue were coincident factors. My patient found she was successful beyond her courage, and becoming alarmed, reported to me. These visions, I think, soon got beyond her control and came against her will, but a cessation of the experiments and a little hygienic treatment stopped them.

genesis from those occurring spontaneously in normal people, and, there are good reasons for believing, may be the product of the same psychical laws as are some hallucinations, at least, of the insane. It may therefore be inferred without much danger of error, that a study of artificial visions will throw light upon the genesis of the spontaneous variety. The experimental method in favorable subjects allows us to study the phenomena with a fair degree of method, and to so alter the conditions as to vary the etiological factors.

It is to be hoped that further work will be done in this field. I have had an opportunity to make some experiments of this kind on a particularly favorable subject. In most of the observations reported hitherto, it has not been possible to thoroughly investigate the relation of the vision to antecedent events in the subject's life, beyond the evidence of the waking memory of that person. But in this instance, the fact that it was possible to hypnotise her and obtain *two additional and distinct personalities, three in all*, each with distinct memories, gave an opportunity to search in the hidden depths of consciousness, and obtain information about facts long forgotten by the normal personality. Further, it was possible to experimentally study the relation of the sub-conscious personality to, and the influence of this upon, the production of visions.

To understand this it is necessary to state a few facts regarding the subject. Miss X. suffers from what is ordinarily called neurasthenia, or hystero-neurasthenia, but what I think is more correctly called hysterical neurasthenia. But she has no physical stigmata, excepting a possible contraction of the field of vision.[1] She is easily hypnotised; first passing into a state which resembles that of the ordinary classical deep stage. After waking there is complete loss of memory of this state. During hypnosis she is very susceptible to suggestion; but on the whole there is nothing very remarkable about the mental condition, which does not materially differ from one of the deeper stages of hypnosis as ordinarily observed. The only points to which

[1] At the time these observations were made this seemed to have disappeared, or, at least, could not be recognised by the finger test.

I would call attention are that Miss X. in this stage of hypnosis knows all about Miss X. awake, and apparently remembers all that Miss X. remembers and some other things besides, which she has forgotten when awake, but Miss X. when awake knows nothing of what has occurred in this hypnotic state. For convenience' sake, I have called Miss X., in this stage of hypnosis, X. 2, as distinguished from Miss X. or X. 1.

Now, on commanding X. 2 to sleep more deeply, there appears a third personality, whom I have called X. 3. This person knows all about both Miss X. and X. 2, everything they do and think, but is unknown to both. Her memory, also, in some respects, is much fuller than that of either Miss X. or X. 2, so it comes about that X. 3 can tell much in the past life of Miss X. that that person has forgotten, and can explain much that the waking personality is at a loss to account for. X. 3 even knows all about many of the little absent-minded and half-voluntary doings of Miss X., nor does she hesitate to voluntarily tell of them, although Miss X. is morbidly and unnecessarily reserved about her whole life.

I have been enabled to record a large number of interesting psychical phenomena which were exhibited by this patient, and which I shall report at another time, but the above facts are all that it is necessary to mention in this connection. It may only be added that the characters of the three personalities were very different and distinct. Miss X. is reserved, morbidly conscientious, self-contained, serious, differential, and dignified. X. 2 is sad, serious, and gives the impression of weariness and suffering. X. 3 is flippant, jovial, free from all physical infirmities, full of fun, reckless, and contemptuous of Miss X., whom she stigmatises as silly, stupid and dull, and in an apparently heartless way enjoys every trouble that comes to her. X. 3 always speaks of Miss X. as "She,"[1] and insists that they are different persons—that they don't think the same things or know the same things. She speaks of X. 2 as "Miss X.

[1] In the following account, She, with a capital S, always refers to Miss X (X. 1).

asleep." This much will explain the conditions under which the following experiments were made. A study of the results obtained shows that the visions could be divided into three groups.

(*a*) Those which were revivals of past visual experiences, either conscious or unconscious.

(*b*) Those which were not revivals, but largely newly created visual representations, of a past experience other than visual.

(*c*) Those which were neither revivals nor representations of any past experience (visual or other) so far as was known.

Naturally some visions partook more or less of the characteristics of two or more groups, being partly revivals and partly new creations; nevertheless, all could be placed in one or other group according to the chief characteristic.

(*A*) *Experiments in which the visions were revivals of past visual experiences*, either conscious or sub-conscious.

Experiment 1.—In this and all of the experiments an incandescent electric light globe or lamp was used instead of the ordinary glass ball. Of course the bulb was not connected with the electric wires or circuit, but held free in the hand of the subject as if it were a mirror. This was the first experiment of this kind made with the subject, and in fact the first I had ever made. The idea of making the experiment only occurred to me at the moment, and I picked up the lamp which was lying on my desk without previously having given any intimation of the nature of the experiment to Miss X., who afterwards told me she had never heard of such phenomena. She was not, therefore, forewarned, and had no idea what was going to happen beyond that suggested by my first remark, which was that she should look into the glass globe, and she would see some thing in the past or future. Nothing specific as to what she should see was suggested. Miss X., after looking a few seconds, exhibited distinct evidences of fright, so that she kept taking her eyes off the bulb to avoid seeing, and obliged me to insist upon her looking. She afterwards explained this fright as due to a feeling of uncanniness. As she looked she described to me what she saw. She saw a young woman in a low-necked dress, blonde hair, about twenty-three, sitting in a chair near what was apparently a chimney-piece

in a strange room. She was laughing. Standing in front of her was an older man with a dark beard tinged with grey. He was scolding her hard, but the young woman was laughing. Miss X.'s face during this time was expressive of very lively emotions, and I was obliged to calm her and assure her that it was all right, and there was nothing to be afraid of.

Here I may mention, once for all, the objective characteristics of these visions as they appeared to the subject and were described by Miss X., and also Miss X.'s mental condition at the time as I observed it. The visions were not seen like small objects reflected in or on the glass bulb, but Miss X. stated that the bulb disappeared, and she saw before her the scene she described, which appeared real, the characters being life-size and like living persons. I should infer that she saw them much as one sees the characters on the stage of a theatre, but she saw them, nevertheless, where the bulb was. Perhaps the illustration of looking through an opera-glass at the stage is apposite, for she several times at first broke off from her inspection and examined the bulb to see if there was any explanation of the vision to be found in the glass, much as one who had never seen an opera-glass or a kaleidoscope might examine it for this purpose. To me as I observed her she appeared like one who, at a theatre, was completely absorbed by the play, and in that sense was unconscious of surroundings, but not at all in a trance state. Her absorption and the exceeding mobility and expression of her face when describing a vision gave the impression that she was entirely oblivious of myself and surroundings, until spoken to, but not as one hypnotised, but rather as one who is intensely absorbed in the scene of a theatre and has forgotten where she is. Every feeling, timidity, surprise, interest, seemed to be expressed by the play of her features, and at times, especially at first, she seemed rather frightened by the uncanniness of what she saw. Most of her descriptions were given in answer to my questions, which were continuous but never suggestive. They were simply expressions like "What else?" "Go on," "Is there anything more?" &c. It may be noted, then, as psychologically of interest, that the visions appeared like ordinary hallucinations or vivid dreams, the scenes real, of life-size, but *dissociated* from her surroundings, and not as part of them.

To return, Miss X. said she could not see anything more. (It was not noted whether the vision cleared spontaneously.) Upon being interrogated she could give no explanation of the scene. She could not remember ever having seen the man, or the girl, or the place before. I insisted that the vision must have

been a part of her past experience, but she could not recall anything like it to memory. I then hypnotised her and X. 2, the second personality, appeared. X. 2 also said she could not remember having seen either of the characters or the room before. She was then placed in deeper hypnosis, and the third personality, X. 3 appeared. X. 3 immediately, as soon as present, began to smile, then burst out laughing, and at once said she recollected the whole incident. It was at San Antonio, Texas, about seven years ago, when "She" was sixteen years old. They had left the dinner table, she said, and were going upstairs to their room, and on the way passed by an open door of a room. In the room were this young woman and the man, and he was scolding away, and she did not seem to mind it. They were strangers, and "She" only saw them once afterwards, driving in a carriage. X. 3 was highly amused at the recollection and the experiment. She was then awakened, and I told Miss X. (without giving the source of my information) that she had seen these people in San Antonio, Texas, and gave the particulars as above. She still failed to recollect the incident. The next day, however, she returned and said that after going home she had gone over in her mind her experiences in San Antonio, and by tracing successively in her mind the different events that had occurred there she had succeeded in bringing back into her memory the whole incident of the crystal vision. In answer to my objection that her recollection was an hallucination and was merely what had been suggested to her by me, she gave a great many more details preceding and following the event of the vision, showing that her memory now is much fuller than that which was shown in the vision. The scene in the room was a mere casual observation, and was not apparently of a nature to make an impression at the time, excepting that it produced some sort of unpleasant feeling, which she always feels when she hears people scolding. I noticed that she manifested some mental discomfort when she described the man in the vision scolding.

The conclusions to be drawn from this experiment are: First, that in this class of cases the hallucination represents a past visual experience—a visual picture was reproduced as a whole. Secondly, that the hallucination is in association with certain sub-conscious groups of mental states or personality (X. 3), but is not in association with all sub-conscious groups (X. 2). Whether it is more than in association with the group X. 3, that is an integral part of the sub-conscious

group in the sense that it was X. 3's vision, the experiment does not allow us to determine. It is odd that of all the experiences of her past life such a trivial one should have been selected to emerge from out of the depths of subconscious life into consciousness. The continuous action and succession of events in this and other visions should be noted.

Experiment 2.—Miss X. looked into the globe and was highly amused. She saw Dr. C., first in a room of the Massachusetts Eye and Ear Infirmary, and then walking down the corridor. She told me that she had seen him there, but never doing what he was doing now. He was fidgeting about in the most nervous way, and walking down the corridor in a funny way. She insisted that although she remembered seeing him under the same circumstances as in the vision, she never saw him acting in that way.

Hypnotised.—X. 3 is full of fun to-day and rather rollicking (on this day experiments Nos. 3 and 6 were also made); told about the visions and enjoyed the thought of them hugely; spoke with great rapidity, rattling them off as if she were anxious to tell a good story and enjoyed the telling. She said that one day She was in the Infirmary, in the large operating room, with Dr. C. He was on one side of the table and She on the other. He fidgeted and put up one foot on the table and then the other, and moved first one arm and then the other in a nervous way. Miss X. copied everything he did, but not intentionally. Dr. C. became so nervous he could not stand it and ran out of the room. X. 3 laughed heartily at the recollection. This experiment is only interesting as showing how forgotten and trivial experiences may appear in visions.

Experiment 3.—This is an extremely interesting vision, but as it represents a somewhat personal matter, I give only the main facts. Miss X. saw two persons walking down the street. One of these, Y., crossed a field to some rocks by the sea shore, where Miss X. was sitting looking out to sea, with her back to the person approaching. Miss X. was about sixteen years of age, dressed as a young girl with her hair down her back. The approaching person went up behind her. She did not see him at first. He had a wild expression in his face, as if insane. "This," she declared to me, "is not true; he never looked like that." It was very disagreeable to see this expression on his face. She repeated "He looks insane." He took some letters or papers out

of his pocket and tore them up, and crushed his watch, which he had in his hand. He acted wildly. Miss X. kept repeating with some fervour "This is not true," "This is not true." In explanation, she remembered that when she was a girl about sixteen the main incident happened; she did see this person walking across the fields to where she was, but she did not see him approach, and none of the rest was true. He simply came up behind her and spoke to her, &c. He did not do any of the things she saw him do in the vision.

Hypnosis.—X. 3 said it was all true. That She (Miss X.) saw him first walking in the street and across the fields, but that Y. came up close behind her without her seeing him. Then She saw him and spoke to him, but when She turned her face away he looked fierce, and crunched his teeth. He had his watch in his hand, and crushed it. X. 3 insisted that She did not see this, did not see the expression of his face, because every time that She turned round he smoothed his face out, and when She turned away her head this fierce expression came again. When I asked X. 3 how she could see that if her head was turned, she kept saying, "I did see it, but She didn't. She doesn't know. I saw it, and that he looked that way. She doesn't know he crunched his watch and tore the papers." (It would appear probable, assuming all this to be literally true, that Miss X. must have caught glimpses of this out of the corner of her eye, making use of peripheral as distinct from central vision. These glimpses were not distinct enough to make a conscious impression, but were seen by the hypnotic self, X. 3. She may have heard the crunching of the watch and of the papers. It is more than probable that the details in the crystal vision are largely exaggerated, being created unconsciously out of the inferences which Miss X. or X. 3 drew from what she saw, a peculiarity of these visions, which is illustrated by some of the other experiments. X. 3 probably didn't see as much as she thinks she did, but woman-like, gives her inferences instead of her actual observation.

Experiment 4.—X. 3 now (after explaining the last vision) volunteered the following remarkable story, telling it with great gusto and as a joke on Miss X., and speaking with great rapidity, so that it was difficult to follow the sequence of events. The language was substantially that of X. 3, but condensed. "She" yesterday received a letter from a photographer. She had it in her hand while walking down Washington Street, and then put it into her pocket (side pocket of jacket) where She kept her watch and money (bank notes). Then as She walked along She

took out the money and tore it to pieces, thinking it was the letter from the photographer. She threw the money into the street. As She tore up the money She said to herself "I wish they would not write on this bank-note paper." At my request X. 3 repeated the words of the photographer's note, which was to say some photographs were ready. As to the money, there were twenty dollars, two ten dollar notes; this, at my demand X. 3 counted mentally, with some difficulty and concentration of thought. X. 3 manifested considerable unwillingness to show me the letter, which she said was in "her" pocket still, and which She still thought to be money. Finally, after some insistance, she did so. It was folded up into a small square, just as one often folds bank notes. The language of the photographer's note was identically the same as quoted by X. 3. X. 3 said that "She" was absent-minded, and thinking of something else, when She tore up the money. I then gave X. 3 the note to put back in her pocket preparatory to waking her up. This impish, hypnotic personality remarked upon what a joke it would be when She found it there instead of the bank notes. I will say here the heartless, cold-blooded delight which this almost Iago-like personality found in the loss of the money, which was a very great one to Miss X., was appalling. To X. 3 it was a splendid practical joke.

Miss X. was now awakened. I asked her whether she did not have some money and had not received a letter from a photographer. She said "Yes," but seemed to think it rather queer my asking these questions, but by this time she had become accustomed to being astonished. In reply to a series of questions she said she had not the letter with her, but had torn it up and thrown it into the street (Washington Street). The bank bills she had in her pocket. They were two ten dollar bills. I asked her to show them to me. She put her hand in her pocket to take them out and brought out the photographer's letter. She evidently received a shock although she tried not to show it. I asked her where the bills were, and after searching her pocket she insisted that she must have left them at home. I remarked that she must have destroyed them by mistake instead of the letter; she refused to admit it. I pointed out the circumstantial evidence; she recognised that it was suspicious but could and would not believe it. The loss meant a great deal to her, and she evidently encouraged herself with a forlorn hope. I then said, taking the glass globe, "We will see whether it is not true. Look in and you will see what you have done." At first she saw only indifferent things; then I said, "Think of

bank notes and the feeling of tearing them up." Now, to her astonishment, she saw herself walking along Washington Street and putting the letter in her pocket, then taking out what looked like bank notes, that is green pieces of paper, and tearing them into pieces and throwing them into the street. The vision in all its details corresponded to the account given by the hypnotic personality. Miss X., in answer to my question, said she had once before done a similar thing, namely, in a state of absent-mindedness, she had thrown some bank notes into the fire and had taken the letter in which they were "down town" to pay some bills. The next day Miss X. reported that she was unable to find the money at home, and was satisfied the vision was true. In hypnosis, X. 3 now volunteered the information that Miss X. ("She") was so much upset by the loss of her money, that in the middle of the night She "had to get up" in her sleep, without knowing it, and had taken the remainder of her money and hidden it under "that floppy thing" on the table. "It was now under a red book, a blue book, and that floppy thing" (by this is meant either a table cloth or a folded up piece of material). "She" knows nothing about it, and thinks She has lost the money and has none left. X. 3 does not know how much money there is. She is very reticent, does not like my knowing, and will not admit the fact. (To have this understood it would be necessary to go into a rather lengthy explanation of the circumstances of Miss X.'s life, which I hardly feel called upon to do. I will merely say that Miss X. had come to Boston to be under my professional care and was in lodgings and had a certain fixed sum for her expenses; the loss of this money meant financial bankruptcy.) Without further discussion, and *without disclosing my knowledge*, I then presented a glass globe to her; told her to think of the money and she would see what had become of it. She looked into the globe and saw herself in bed in her room. She then saw herself get up, her eyes being closed, and walk up and down the room; she then saw herself going to the bureau drawer, taking out her money, going to the table, taking up the table cloth with the books, putting the money on the table, covering it with the cloth and putting the red book and the green book on the top of it. The vision thus exactly corresponded[1] to the statement of X. 3. Miss X. reported at the next visit that she had found the money where she had seen it in the globe.

[1] Excepting the colour of the book, "blue" or "green." This escaped my attention at the time; perhaps it was blue-green, and looked blue to X.3 and green to X., or it may be an error on my part.

Remarks.

It is noteworthy that in this experiment, as well as the preceding one, the vision was a representation of the hypnotic or subliminal consciousness, and not of the waking consciousness. In experiment No. 3, certain things were portrayed in the vision which the waking consciousness (apparently) never perceived, while in experiment No. 4 (first part, destruction of bank bills) an absent-minded act was represented, and in the second part a somnambulistic act. It might be held that the first vision of the bank note episode might have been created out of my statement to her, if it were not for the minute fidelity with which the vision corresponded with the account given by X. 3, and the corroborating evidence of the second vision when I gave no hint of what would be seen.

(*B*) *Experiments in which the hallucination was not a revival of a past visual experience, but was largely a newly created visual representation of a past experience other than visual.*

Experiment 5.—I will give this in the brief language of my notes: Miss X. looked again into the globe; she saw a room with a bed in it. There was a figure in the bed; the figure threw off the bed clothes and got up. Miss X. exclaimed "Why, it is I." (Appeared rather frightened at what she saw, but went on to describe it, largely in answer to my promptings, such as "Go on," "What do you see?" &c.) She saw herself walking to and fro, up and down the room. Then she climbed on to the window sill which is the deep embrasure of a mansard roof. Then she climbed outside the window and from the sill looked down into the street. It was night—the street lamps were lighted, there was also the gas light in the room. As she looked down she felt dizzy. Here Miss X. turned away frightened, saying she felt the same dizziness as if she were standing there. She soon continued. She saw herself throw an inkstand into the street below which she had seen herself take before climbing on to the window. Miss X. was again obliged to stop looking because of dizziness. After a time she returned to the globe. She saw herself go back into the room and walk up and down; the door opened and she jumped into bed and lay quiet. Miss L. (a friend) entered, went out and returned several times;

brought a poultice which she put on Miss X.'s chest; Miss X. herself remaining quiet. Then Miss L. went out and Miss X. got up and took the poultice, rolled it up into a little bunch and hid it in a corner, putting a towel over it. Here the experiment ended.

Miss X. stated, on being questioned, that she could not remember any incident like the vision, excepting that she recognised the room as the first one she occupied when she came to Boston four or five years ago. It was in the top story of a house on Columbus Avenue; she was ill there, and Miss L. took care of her. But she did not remember ever having climbed on to the window, or having thrown an inkstand, or any of the incidents of the vision. She could throw no light on the affair. She was now lightly hypnotised and X. 2 was present. X. 2 could add very little to Miss X.'s statement. (My notes of X. 2's memory are somewhat confused; it is not plain from them whether or not she remembered ever having done any of the things seen in the vision, although it is stated that she had an imperfect memory of the incident. It is possible X. 2's statement as noted was mixed up with that of X. 3, but X. 2 remembered being ill in the room described, and that ink was found in Miss X.'s shoes; X. 2 did not know how it came there, but Miss L. had said that Miss X. had poured ink in her shoe. At any rate X. 2 could not explain the incident as X. 3 did later.)

Deep hypnosis; X. 3 appeared. With great vivacity and amusement X. 3 explained the whole scene. (As was customary with her, X. 3 spoke of Miss X. (X.) as "She," as if it were an entirely distinct person and not herself.) "She" had pneumonia and was delirious, and She imagined She was on the sea shore and was walking up and down the sand. This was why She walked up and down the room, and She stuck her toes in the carpet thinking it was the sand. There were rocks there, and the window sill was one of them, and when She climbed out upon the window sill She thought She was climbing upon a rock, and She took up a stone, as She thought, and threw it into the sea. This was the inkstand that She threw into the street. Then when she took the poultice and hid it in the corner She thought She had buried it in the sand. She had not poured ink into her shoes, but her hand shook and She had spilled it into her shoes. Miss L. seeing the ink stains had inferred that Miss X. had poured the ink into the shoes, and had told Miss X. so. Hence the statements of X. and X. 2. X. 3 was highly amused at all the mistakes of Miss X.'s delirium.

Remarks.

It is obvious that this vision was not a revival of an optical experience of the past, for Miss X. could not have seen herself as she did in the vision. The hallucination was rather a new creation corresponding to, and illustrative of a memory of, a past event. But into this new creation a certain number of past optical images entered, as in the scene in the street and the room. Yet even these were originally seen by the delirious consciousness in a different way, that is as if it were the sea shore. The vision rather corresponded to the way in which the third personality, X. 3, would have seen delirious X. 1 and her surroundings if she could have seen them. This suggests the relation of the hypnotic personality, X. 3, to the delirious consciousness. X. 3 observed the delirious Miss X., and was cognisant of her thoughts just as she was of those of the normal Miss X. Just as X. 3 knew all about X. 1 when in health, so she knew all about X. 1 when delirious. But normal X. 1 had no recollection of delirious X. 1. The hypnotic consciousness is not the delirious consciousness, but bears the same relation to it that it does to the normal consciousness. X. 3 was normal while X. 1 was delirious. It is a startling idea, that of there being beneath the delirium of a person in fever another healthy personality, a mind unchanged, which is observing everything done, thought and said by the delirious person. It is worth noting in passing that when Miss X. saw herself looking into the street, she at that moment actually felt dizzy. That is, as the scene appeared real to her, looking down into the street of the vision made her dizzy.

Experiment 6.—This experiment was interesting, as it showed how a vision could be constructed out of certain past familiar images and certain other experiences of which no optical images could have been received; a new synthesis of images being created by the force, if I may use the word, of imagination and known facts. Looking into the globe, Miss X. was astonished to see herself sitting on a sofa, the identical sofa on which she was then sitting. *Her eyes were closed*, and she was smoking cigarettes. While Miss X. was looking at herself in the globe she

several times turned to look at the sofa on which she was sitting to compare it with that in the globe. They were the same. Her whole expression was one of extreme astonishment and chagrin. She indignantly repudiated the fact, said it was not true, she was was not in the habit of smoking cigarettes, and had never smoked one. In giving a further description of herself in the vision she said her eyes were closed and she looked foolish. The explanation of this vision was that the day before, as a practical joke, I had given a cigarette to X. 3, who had taken it in a spirit of fun and smoked it clumsily, showing plainly she had not been accustomed to smoking. X. 3 laughed and made merry over the prank, saying it was a great joke on Miss X., who would be "awfully shocked" if She knew it. Miss X. pretended it was very wicked—as it was for her. The visionary Miss X.'s eyes were closed, for I had always obliged X. 3 to keep her eyes closed for reasons not necessary to go into here.

Remarks.

It is noteworthy that the vision, so far as it concerned cigarette smoking, represented an experience which belonged only to the hypnotic personality of which Miss X., the superior consciousness, never had any knowledge. From this it might be inferred that visions might represent absent-minded acts as in the case of experiment No. 4. Secondly, the contents of the vision were partly previous optical images received by the primary consciousness, that of Miss X., viz., images of the sofa and portions of Miss X. (X. 3 had never had her eyes open), and partly were syntheses of the third personality's sole *knowledge;* thus the vision was made up of the experiences of two personalities, and only in part of past images. As she had never *seen* herself smoking, the vision was largely an optical representation (not a revival) of impressions from other senses.

(*C*) *Experiments in which the vision did not reproduce or represent any past experience, visual or other, so far as was known.*

Experiment 7.—Miss X. looked into the glass globe. She saw an old lady standing in a very steep street leading up over stones; there were steep rocks on one side. The street was like nothing

she had ever seen before in this country. (She said *this country*, as if to emphasise the fact that it looked foreign.) She had never seen the old lady before. As she looked, a man on horseback appeared, and the horse knocked the old lady down. The latter seemed to be hurt. Here Miss X. turned her head away as if alarmed. On another day Miss X. again saw the same vision. The man's features were also strange to her. The most minute inquiry failed to elicit any fact that would throw light on this vision. Miss X. said she had never been abroad, and had never seen any place resembling that of the vision, nor knew of any such accident. When hypnotised neither of the other two personalities (X. 2 and X. 3) had any recollection of any such place or event. I am disposed, therefore, to believe that it was probably a visual representation of something she had read, or of some picture she had seen, and which would readily pass out of the recollection of even the hypnotic self. As she described the street in detail it recalled places to me I had seen in Europe, or pictures of foreign scenes.

Experiment 8.—Miss X. had lost a scarf pin she valued. I suggested that she should try to find it by looking in the globe. After looking a few minutes, she saw a bed-room with a brass bedstead, and on the dressing-table was a pin-cushion (described in detail), and in the pin-cushion were several pins. Amongst them she recognised her own, and exclaimed somewhat excitedly "Why, that is my pin!" Involuntarily she reached out to seize it. During this her face was very expressive, and showed great astonishment. She did not recognise the room, and was certain she had never seen it before, but thought it a city room and not a country one from its general appearance and furnishing.

In hypnosis both personalities were also ignorant of this room, but X. 3, being asked to tell the last time that Miss X. had the scarf pin, recollected perfectly. "She" (Miss X.) came into her room (in the country, not in Boston) one day with a very severe headache, took the pin out of her dress, and stuck it into the head of the couch, then doubled up the pillow and placed it over it, and lay down on the couch with her head on the pillow and rested. "She" forgot all about the pin, and had no recollection of putting the pin where She did, and, in fact, did it rather absent-mindedly. This was several months ago, and X. 3 presumed the pin was gone. On Miss X. being awakened, I told her what she had done with the pin, but still she had no recollection of the affair.

Remarks.

Probably in this case the vision did not correspond to any known fact in Miss X.'s experience. The room, I suppose, might be some room she had casually seen but forgotten, but she could not very well have seen her pin in it. Perhaps she might have thought, at some time, that the pin had been found or taken, and was in the possession of someone. Thus the vision might have been constructed in much the same way as the cigarette one (experiment No. 6). I was unable to learn whether X. 3's story was correct or not, but it probably was, as I had always found her positive recollections accurate. This experiment also illustrates, like No. 4, the effect of suggestion in determining the character of the vision; that is, a vision of a particular thing or subject in preference to any other vision. I told Miss X. she was to look into the globe for information of the pin, when she saw the contents of X. 3's consciousness as a vision; in other words, it was not her own subconscious personality that came up from below into consciousness as a vision, on the contrary, it was X. 3's proper consciousness. This seems also to have been the case in experiments Nos. 3 and 6.

I wish that further observations had been made on this subject, especially with a view to determining how far the contents of a vision could be shaped by suggestion, and the relation of visions to beliefs, and other thoughts; but various considerations made it desirable to interrupt the experiments at this point. As the laws governing one sense are probably the same as those for another, it is likely that the antecedent relations of hallucinations of hearing are the same as those of hallucinations of vision. This inference is somewhat speculative, but it gives additional value to a knowledge of the origin of visions. If it be true, as is most highly probable, that visions may be an optical representation of pure thoughts and beliefs, this fact gives us something with which to answer Griesinger's question, "Why does the insane patient believe in his hallucina-

tions?" It would seem as Hirst [1] well says, "The patient does not believe himself to be the son of the emperor, because the voice told him so, but he heard the voice making this statement because he believed it."

Conclusions.

(1) Visions in sane persons may be revivals of past visual experiences, which originally may have been conscious or sub-conscious. The original sub-conscious experience may have occurred in a moment of absent-mindedness, or may not have been sufficiently intense to have entered consciousness, or (rarely), may have occurred in somnambulism.

(2) The vision instead of being a revival, may be a newly-created pictorial representation of a past experience other than visual. That is to say, past impressions of one or more senses (touch, hearing) and actions may translate themselves into a representation by another sense as a vision.

(3) It is probable, though not proved, that a vision may not reproduce or represent any past experience, visual or other, but may be newly created out of something the subject has read, heard, or thought. The inference from, and passing thoughts about, known facts may weave themselves into visions. This was probably the origin of the visions of Joan of Arc, and religious enthusiasts.

(4) Visions may partake more or less of the characteristics of these classes, being partly revivals, partly representations of actual non-visual experiences, and also of the subject's knowledge, inferences and thoughts.

(5) Generalising, it is possible that hallucinations of the other senses, especially of hearing and as exhibited by trance mediums, may have a similar origin and composition.

(6) Analogous phenomena may be observed in the attacks of hysterics where the passing thoughts in the normal state may appear as insistent ideas in the attack.

[1] "The Psychical Mechanism of Delusions." The *Journal of Nervous and Mental Diseases*, March, 1898.

(7) It is probable that thoughts which have strongly absorbed the mind and expressed the longed-for ambition, or the ideas and beliefs of the subject, may appear as visions. The subjects then are apt to look upon them as inspirations. In this way have arisen the visions of political personages like Joan of Arc, Bismarck, and religious enthusiasts like Luther, Fra Angelico, Catherine of Sienna and others.

(8) Visions, artificially created, may be representations and revivals of the experience of the hypnotic personality, of which experience the waking consciousness has never had knowledge.

(9) Impressions on the sense organs which never entered consciousness (therefore neither known nor remembered), may afterwards appear as visions.

A CONTRIBUTION TOWARDS THE DETERMINATION OF THE ENERGY DEVELOPED BY A NERVE CENTRE.[1]

BY VICTOR HORSLEY, F.R.S., F.R.C.S.

I HAVE first to return my very sincere thanks to the Society for the honour that they have conferred upon the surgical side of Neurology and upon myself in electing me to their presidential chair, and I appreciate the honour the more since it is a position which has been occupied successively by almost all my teachers, prominently among whom is my immediate predecessor, Dr. Savage.

It is the established custom for the person just inducted into the chair to deliver an address, and I am very glad to embrace this opportunity of communicating, if in a somewhat preliminary manner, the results of an investigation which I planned some years ago, and upon which I have also worked from time to time partly in collaboration with Dr. Veraguth and Dr. Christiansen.

The subject matter of this research is an attempt to estimate quantitatively the "work"[2] done by the central mechanisms of the central nervous system when these discharge their nerve energy; for example, to discover, if possible, the amount of energy developed by the centres in the cortex cerebri and in the spinal cord respectively, the relative volume of the two mechanisms being so very different, see fig. 1. Apart from the general scientific interest of prosecuting this study of nerve energy for its own sake, this subject I venture to think has a considerable

[1] Presidential Address to the Neurological Society.

[2] Of course the word "work" is not used here in its scientific sense, since we do not know the nature of nerve energy or nerve force. The words "energy" and "force" are also to be accepted in their popular meaning.

importance in relation to the epileptic convulsion, and therefore I bring it forward to-night as a matter of concern to both the physiologist and the clinician.

When we speak of the discharge of nerve energy from a nerve centre it is not a little disturbing to find that we know nothing about nerve energy except the rate of its transmission along the nerve fibres and the circumstances under which it is evolved. It has of course been compared

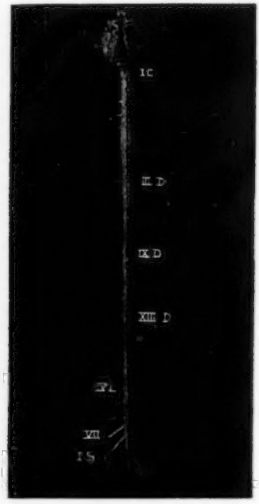

FIG. 1.

Central Nervous System of the Cat.

to all forms of so-called physical energy, and Newton himself speaks of a nerve impulse as amounting to a vibratory disturbance of the particles of the nervous system.

Nerve energy cannot therefore be measured with any apparatus known to us at present, and therefore if we are to attempt to study its development quantitatively the only

available method of doing this is by indirect means such as quantitative measurement of either some constant correlative phenomenon or of any physical effect directly produced by its activity. I would suggest that there are two possible ways of arriving at some sort of conclusion: these are (1) electrical, (2) mechanical.

(1) *Electrical Method.*—Of these the first is the measurement of the "negative variation" of Du Bois Reymond or "action current" of Hermann, which accompanies, of course, every single so-called nerve discharge or impulse, and which, in a summated form, may be accurately enough estimated by the galvanometer or electrometer.

It will be realised that the electrical method of examining the degree of discharge of a nerve centre is only the quantitative observation of an electrical change in the nerve channels running from the nerve centre which is the seat of stimulation; in other words, the electrical method offers of course nothing absolute but provides the determination of a very important parallel phenomenon, such phenomenon being developed under such circumstances as make it at least likely that a quantitative relationship exists between it and the discharge of nerve energy. So that in a measure the degree of electrical change may be said to be an expression of the degree of development of nerve energy. The proof of this was given by Professor Gotch and myself six years ago when we published in full[1] the results obtained by our method of investigating the functional activity of various parts of the nervous system. Among our facts we included a table giving the readings of the deflections observed when the galvanometer was connected with the spinal cord and the cortex cerebri excited and when, in addition, the general epileptic condition of the upper half of the body was simultaneously noted. I repeat here a table (constructed from experiments on four different animals) which shows clearly that the electrical change or summated action currents in the spinal cord was quite proportional to the degree of discharge from the cortical nerve centres.

[1] *Phil. Transactions*, vol. clxxxii., p. 331, 1891.

GALVANOMETRIC EFFECTS PRODUCED IN SPINAL CORD OF CAT BY CORTICAL EXCITATION. (*Phil. Trans.*, 1891).

	Intensity of Excitation	Duration and Place of Excitation.	Galvanometric Effect	Muscular Movement
Cat (31) ..	8,000	5″ left cortex	63	Slight fit
	8,000	,, right ,,	115	,, ,,
	9,000	,, left ,,	108	,, ,,
	9,000	,, right ,,	230	Good fit
,, (317) ..	8,000	,, left ,,	50	Very slight fit
	10,000	,, ,, ,,	162	Fair fit
	12,000	,, ,, ,,	235	Good fit
	8,000	,, right ,,	130	Slight fit
	10,000	,, ,, ,,	185	Fair fit
	12,000	,, ,, ,,	230	Good fit
,, (319) ..	10,000	,, left ,,	90	Slight fit
	12,000	,, ,, ,,	170	Fair fit
	10,000	,, right ,,	150	,, ,,
	12,000	,, ,, ,,	260	Good fit
,, (324) ..	6,000	,, left ,,	260	Slight fit
	8,000	,, ,, ,,	340	Fair fit
	10,000	,, ,, ,,	510	Powerful fit
		Average.. ..	17 = 3288	
			193	

I intend to pursue this branch of the subject with the advantage of the use of the capillary electrometer as recently perfected by Mr. Burch, seeing that with the electrical units now in use the disturbance of energy in the spinal cord channels can be expressed scientifically in terms of work and consequently be directly compared with the records obtained from the muscle by the mechanical method *vide infra*. This has been rendered possible by the recent determination by Gotch and Burch, of the true value of the single "negative variation" or "action current." The unsatisfied condition, however, of course, always still remaining to mar this method, namely, that we cannot know the *precise* scale of parallelism between the amount of nerve energy and the amount of the electrical effect, until some one discovers a means of directly recognising and measuring nerve energy.

(2) *Mechanical Method.*—By this expression I wish to convey the fact that advantage is taken of the connection

of certain muscles with certain nerve centres to accept the work done by the contraction of such muscles as representing the degree of nerve energy evolved from the centre the activity of which produces the contraction. For this purpose we require measurement of the "lift," *i.e.*, height and weight lifted, effected by the discharge of any given centre, as well as the total work done in a given time. I shall on this occasion speak of the "lift" accomplished by the muscle and not the areas of the curves obtained in the experiments which would give the total work done. Provided that the centre examined and motor nerve leading therefrom are subjected to a maximal excitation (maximal because since each of these experiments is a comparison and at the same time involves the excitation of structures very different in nature and excitability as nerve centres and nerve channels respectively are, naturally the stimulus must be maximal) then we know that we have reached the

FIG. 2.

Graphic record of Muscle Contraction resulting from Cortical Excitation. (*Phil. Trans.*, 1891.)

limit of mechanical effect obtainable from the part excited, and it then becomes possible for us to compare the effect obtainable from the muscle when directly excited from its nerve with that obtained as the effect of the discharge of nerve energy from the centre. From the comparison of these results we gain a relative value. Whereas in the case of the electrical method there is the drawback, as I pointed out, of want of a precise knowledge of parallelism between the correlated phenomena of nerve discharge or electrical "action current"; in this mechanical method we have a larger and more important source of complication and possible error which must now be noted. If we are

applying the method to obtain an estimate of the activity of the centres in the cortex cerebri, it is a fair assumption that the discharge observed arises in the cortex cerebri, but there remains the exceedingly difficult question as to how far the bulbo spinal centres are, or are not, taking part and producing a certain share of the whole effect. To take a concrete example the question is, given excitation of the centres in the cortex we have to enquire whether the total amount of work done by the contracting

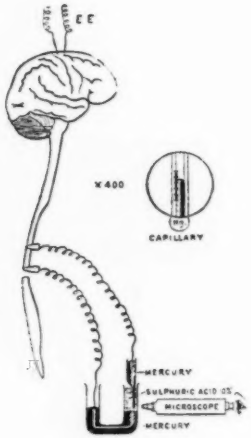

FIG. 3.

Schematic representation of electrometric arrangement for estimation of the descending changes in the cord.

muscle is due to the energy of the cortex, or due to the energy of the cortex plus that of the spinal cord; or, to put the matter yet in another way, the question is whether the spinal cord, under these circumstances, is acting as a mere conductor, or whether it is acting as a contributing agent. As regards the *character* of the contractions of the muscle, *i.e.*, tonic and clonic (see fig. 2), this problem was first satisfactorily approached by Franck and Pitres, who, in 1877, found

by the graphic method that excision of the cortex and excitation of the subjacent corona radiata fibres was followed by practical abolition of the clonic element, so characteristic a feature in the effect derived from stimulation of the cortex. This result was confirmed by Bubnoff and Heidenhain (1881), Schafer and myself (1885). From this fact the conclusion was drawn by some observers that the tonic element was due to the lower (bulbo spinal centres). On this point a controversy arose between Ziehen[1] and Unverricht,[2] which had the effect of emphasising the essentially clonic nature of the discharge of the cortex centres. The

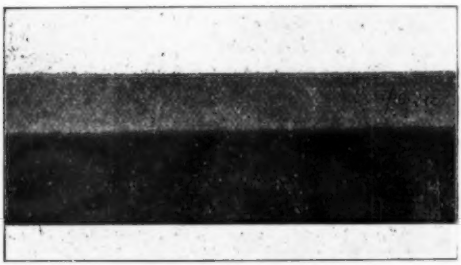

FIG. 4.

Reduced photographic record of the electrometric variations, the cord being connected to the electrometer and the cortex excited.

only proof, however, that could be accepted as really bearing directly on the character of the elements of the cortical discharge was afforded by the electrical method of Gotch and myself,[3] where, by interpolation between the cortical and bulbo spinal centres respectively (see fig. 3), the fact that the cortex discharge consists of *both* tonus and clonus elements was finally established. (See fig. 4 and fig. 5, in which latter the action currents of the clonic spasms are

[1] Chief paper "Zur Physiologie der Infra-corticalen Ganglien, &c." *Archiv f. Psychiatrie und Nervenkrankheiten*, B. xxi., p. 863, 1890.

[2] Chief paper "Ueber Tonische und Clonische Muskelkrampfe." *Deutsches Archiv fur Klinische Medicin.* B. xlvi., p. 418, 1890.

[3] *Loc. cit. supra.*

more highly magnified.) Still there remains the problem, how far in the intact nervous system does the spinal centre add to the work effected, since it is now clear that the *character* of the whole effect is entirely cortical. At present our data are imperfect and must remain so until by the electrical method the work done in the nerve channel between the cortical and the bulbo spinal system respectively has been measured. Now, as regards the mechanical method, I am going to suggest that for the purposes of the present subject this question of the source of the tonus contraction does not stand in our way of

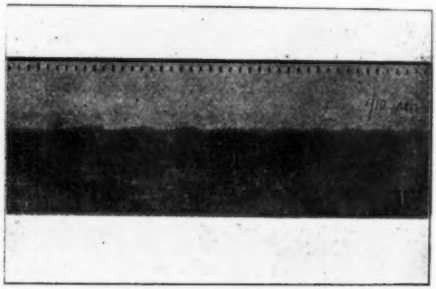

FIG. 5.

The clonic stage in fig. 4 recorded under higher magnification and more rapid rate.

advancing the subject for the reason that it is possible to first obtain a maximal reflex discharge from the spinal centres, and by that means to discover that the cortex cerebri gives a discharge of more energy than that of the cord, or at any rate, during the first few seconds of the stimulation time. In fact, a relative estimation is easy to make when the effect of a cortical excitation is, as a matter of fact, so much larger than that derived from a direct stimulation of the isolated of the spinal cord. Certain further precautions have to be dwelt upon before we can expect this method, which I only put forward as a means

of indicating the relative powers of the different nerve centres to fulfil all we hope from it.

Description of the Mechanical Method.

The mechanical method I have adopted in these experiments is as follows: All the initial operations were performed under ether narcosis. The cortex cerebri was exposed and kept warm. The muscles chosen have been the biceps brachii or the rectus femoris. The latter appears to be the more suitable muscle, certainly it does not become fatigued so soon, or, rather, the exposure of its nerve is not followed by so much shock as in the case of the biceps. An inextensible thread was carried from the muscle over a small pulley and attached to a small metal bucket running on steel guides and carrying a light aluminium marker. The total weight to be raised by the muscle was made equal to 100 grammes by putting shot in the bucket. This, together with three electrical signals, two for recording the moment of stimulation from the two separate induction coils, and the third, a seconds of time marker, was arranged to write simultaneously either on the paper of a Hürthle Kymographion or on the glass plate of Professor F. J. Smith's chronograph, in which the carrier was drawn forward by slow clockwork. Both sciatic nerves were exposed and ligatures passed beneath them, as also the right anterior crural nerve, where it is buried in the substance of the psoas muscle. A slip of indiarubber sheeting was passed under the latter, and fine platinum electrodes were hooked on the nerve in question lying between the folds of the india-rubber. By use of the second coil it was possible to arrange simultaneous excitation of the different structures. After a tracing had been obtained of the muscular spasm resulting from excitation of the cortex, then where possible a tracing was taken consequent upon excitation of the corona radiata, and when the observations upon the cerebrum had been completed, the spinal cord was divided at about the level of the sixth dorsal vertebra, and everything prepared for

direct observations of the reflex movements. These were investigated in the following way:—both sciatic nerves were firmly tied with ligatures and divided peripherally. At the moment of ligature and of subsequent division a tracing was taken. The reflexes were ascertained by first exciting the central end of the left sciatic nerve, the right rectus femoris being always chosen; and if there was any effect obtainable, the direct reflex was determined by excitation of the right sciatic nerve (central end).

Relationship of the Different Nerve Centres.

I have not in the following research attempted to ascertain any influence of the cerebellum whether exerted upon the cortical or bulbospinal centres respectively. Its supposed function as a controller of the cerebrum, surmised theoretically by Gowers and established experimentally by Risien Russell is paralleled by Löwenthal's discovery[1] of its controlling influence on a certain (extension) share of the reciprocal innervation phenomena of the cervical bulbospinal centres. So, too, I have ignored the influence of the basal ganglia, because we do not know what their function is, and the idea that they participate in so-called purposive acts which we now know to be of cortical origin has, I believe, no real foundation in any scientific fact.[2] Moreover, it is not easy to see how they could experimentally be excluded by resection because of the close proximity of the fibres of the internal capsule passing from the cortex down to the spinal cord. The connection between the centres in the cortex and the spinal cord is, of course, of importance in the present question. Topographically it has been shown by the simple excitation method originated by Hitzig and Fritsch and so greatly extended by Ferrier, that the frontoparietal region is alone in direct connection with the centres in the spinal cord, whereas the special sense areas, *e.g.*, occipital, temporal, &c., are in undoubted connection with some of the nuclei of the cranial nerves at the base of the

[1] Löwenthal & Horsley, *Proc. Roy. Soc.*, 1897.
[2] See Beevor & Horsley, *Phil. Trans.*, 1890, p. 49.

brain and bulb. A further proof of this position was also obtained by Gotch and myself[1] by the electrical method, when we found by leading off different parts of the spinal cord to the galvanometer that excitation of other parts of the cortex, *e.g.*, of the occipital lobe, failed to evoke any electrical change in the spinal cord. The nerve centres therefore, which I have chosen for analysis by the mechanical

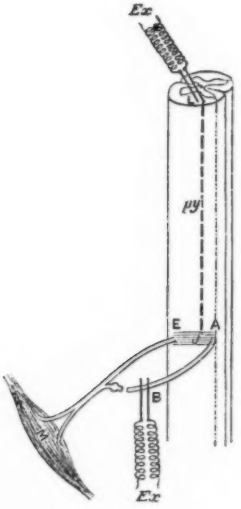

FIG. 6.

Diagrammatic representation of the reflex arc and connection with the pyramidal fibres (*Phil. Trans.*, 1891.)

method are those only of the limb areas of representation in the parietal cortex and the spinal centres for the limbs in the spinal cord.

Narrowing our considerations then to this degree we enter upon the next preliminary question. What is the nature of the direct connection between the cortical centre and the spinal cord centre looking upon the latter as an

[1] *Loc. cit. supra.*

intermediate stage between the cortical centre and the muscle.

The degeneration method has shown that the fibres of the pyramidal system descend from the cortical centres, pass into the spinal centres for the muscles, and therein come in an unknown way, into relationship with the large ganglion cells of the anterior cornua, from which extend an axis cylinder process down the so-called motor root and mixed nerve to the muscle.

By the electrical method Gotch and myself showed that there is a remarkable diminution in the electrical effect obtained from the spinal centres along the large nerve trunks to the muscles as compared with the effect obtained on the spinal cord by excitation of either the cortex or pyramidal fibres of the corona radiata, suggestive of the view that the spinal centre forms an intermediate station of considerable influence (see fig. 6.)

I have thus far dealt with this matter of the connection of the cortical centres and the spinal centres, because it is one of great importance, and because it enters into some considerations later as well, but it does not have a direct bearing upon the immediate *value* of the mechanical method since we find that just as is shown by the electrical method in the spinal cord, so the contraction of a muscle *i.e.*, work done by the muscle, varies directly with the amount of discharge from the nerve centre. As a matter, therefore, of relative proportion the activity of the muscle is undoubtedly a measure of the activity of the nerve centre. This being so we are then able to use the muscle to measure the relative values of the discharge of the different parts of the system. Before taking up the actual result obtained I would like to point out that by the electrical method Gotch and myself found that excitation of the corona radiata evoked an electrical change in the cord, which was slightly more than half that evolved when the cortex cerebri was excited. In this connection I must repeat that there is properly speaking no second stage or clonic after effect when the corona radiata is excited, and that the galvanometric observations are those of summated discharge effects.

So, too, the electrical effect in the nerve when the spinal centres are caused to discharge by reflex excitation give rather more than one seventh of the deflection observed when the cortex is excited. That the pyramidal system of fibres have practically the same degree of connection with the elements of spinal centres, as the fibres of the other channels is shown by the fact that the amount of the galvanometrical deflection obtained from the nerve when the cortex was excited was equal to that noted in the nerve when the discharge of the reflex spinal centre was passing down it.

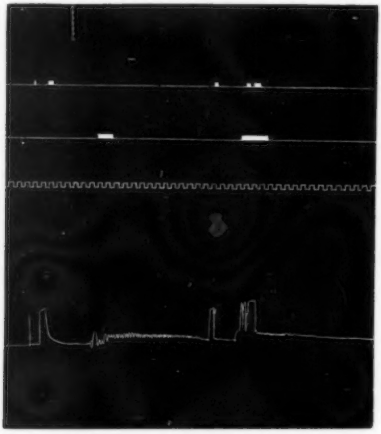

FIG. 7.

Arrangement of the Nerve Centres according to the Degree of Work Done.

In employing as the scale of comparison between the respective heights of the contraction of a muscle in raising the weight of 100 grammes I should state at once that I have never seen any discharge from the nerve centre to result in a contraction of the muscle exceed in height that obtained by stimulating the nerve to the muscle, and in fact no nerve centre by any means that I have employed and

described is capable of evoking as much work from the muscle as excitation of the motor nerve to the muscle produces.

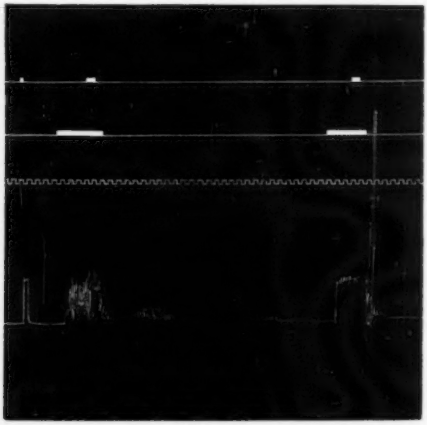

FIG. 8.

FIG. 9.

Figs. 7, 8 and 9.—These figures show the varying degrees of "lift" obtained from the cortex cerebri and motor nerve respectively and in association.

Fig. 7.—In this and succeeding photographs of tracings (reduced), the uppermost line marks by an electric signal

the moment and duration of excitation of the sciatic nerve; the second tissue similarly marks the excitation of the cortex cerebri, and later the spinal cord and central end of one sciatic nerve; the third line marks seconds; and the bottom line is the record of the weight attached to the muscle.

In other words, the nerve centres do not cause a complete explosion of the potential contractility of the muscle. Further, the average "lift" derived from the cortex is about three-fifths of that obtained from excitation of the motor nerve.

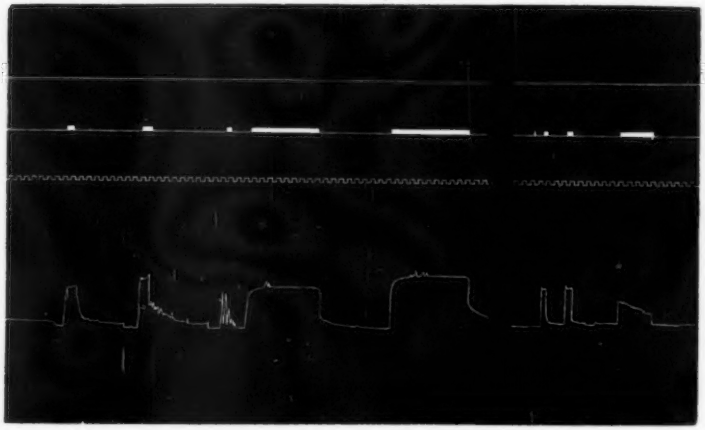

FIG. 10.

In an experiment conducted for the purposes of comparison such as I have described the cortex cerebri is at an advantage compared to the spinal centres, because clearly the order of events must be that to test the effect of the centres respectively on the same muscle the cortex phenomenon must be observed before the cord is cut for the purposes of obtaining in its pure form the discharge from the spinal centres. Thus the spinal centres in such an experiment are at great disadvantage, inasmuch for a time they are depressed by shock; but in this connection the hyper-excitability which soon supervenes and which was

first minutely described by Franck,[1] counterbalances the error if the excitation results are taken at proper times after the section of the cord. To obviate this I have varied the experiment in different ways.

Allowing for errors in complications just referred to it would appear certain that the discharge from the cortical centre is on the average capable of evoking more work from the contracting muscle than a spinal centre, so that the centres should be arranged in that order cortex first and spinal cord second.

Thus I have performed some experiments in which the spinal cord was divided first, therefore no cortical excitation was undertaken, and only the spinal cord discharges examined with the result that a higher average "lift" was obtained.

Differentiation of the Effects.

(*a*) *Cortex or Cortical Effect.*—The character of the cortical discharge I have referred to, but I would like to point out that even at the present time the analysis of its components is very incomplete, and now propose to enter at once upon its analysis.

Firstly, the amount of energetic action by the centre, *i.e.*, demonstrated by the degree of the contraction of the muscle, varies inversely as Bubnoff and Heidenhain,[2] originally showed with the rapidity of the discharge of the centre, or, putting it in other words, with the shortness of the latent period. This fact is the experimental proof of Dr. Jackson's theoretical conclusions[3] on this subject.

In taking the whole curve of the muscle contraction from the cortical discharge we see at once that the greatest amount of work is performed as a rule shortly after the commencement of the excitation, that is, at the beginning of the fit; but this varies with the maximal or minimal character of the excitation. A minimal excitation produces a slow discharge of energy from the nerve centre (see fig. 11), and in accordance with the principles just stated resulted in a less

[1] *Loc. cit. supra.*
[2] *Archiv. f. d. Gesammte Physiologie.* B. xxvi., 1887.
[3] "Lumleian Lectures," *Brit. Med. Journal*, vol. i., 1890.

amount of work being executed early by the centre; whereas the maximal stimulus produces a high tetanic curve from almost the first moment, which is maintained at the same level for a short time, and then in respect of the main-

FIG. 11.

tenance of the application of the stimulus to be discussed presently gradually sinks towards the abscissa line. The moment the stimulus ceases, after a rapid diminution usually to or near the abscissa, there occurs a secondary

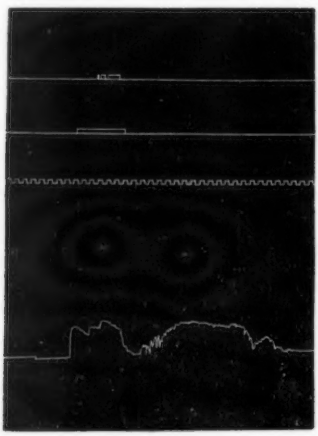

FIG. 12.

gradual development of work done, and very rarely this after effect may amount to nearly the height obtained by the primary discharge from the cortex, *i.e.*, of the first stage. (See fig. 12.)

The clonic or secondary stage of the cortical effort usually attains to a certain height practically equal to that of an initial contraction, at least so far as the apices of individual clonic spasms, and this has been looked upon as a direct result of cortical activity. The character of its tetanic average is that of a gradual rise and an equally gradual fall.

The original experiment of Franck and Pitres, showing that when the cortex is cut off and the subjacent fibres of the corona radiata excited there is no clonic or secondary stage, has always been accepted as indicating that the clonic stage is entirely cortical, but actual proof was wanting until as stated above, the electrometer tracing showed that the clonic spasms were innervated by intermittent impulses of a similar rate and degree descending the spinal cord. It is now, therefore, clear that the clonic factor is of cortical origin, but the question remains, What is the source of the work done in the tetanic reinforcing curve of the clonic stage, is this also cortical or of spinal origin?

I have for some time been investigating this in relation to the whole subject of work done by nerve centres, and by varying the experimental procedure hitherto adopted have obtained some additional results.

It seemed to me that if there was this doubt, it could be to a certain degree cleared up if we could exaggerate the phenomenon itself, *e.g.*, by raising the excitability of the whole system, and especially the spinal cord. For this purpose in a certain series of experiments I stimulated, as shown in the tracing, the central end of the sciatic. Bubnoff and Heidenhain found that powerful excitation of the central end of a sensory nerve caused an increase in the amount of "nerve energy" put out by the cortex.

This we shall see is not always correct, and the exception reveals a very important condition of things.

In repeating these experiments in a slightly modified way I found that the tetanus of the *clonic* or secondary stage was increased when the ordinary tracing was taken and at last obtained in one striking instance the accompanying tracing (fig. 13), which shows what I wish to call a

typical spinal cord effect occurring *after* the clonic stage, showing that at any rate in this instance if the spinal cord was in any degree responsible for the tetanus of the clonic stage it must be to a small amount. A similar tracing was obtained and figured by Franck,[1] but is not explained by him.

At this point I think it would be better to take up the question of the discharge from isolated spinal centres.

Discharge from Spinal Centres.—To compare the maximal discharge from the spinal centres with that obtained from

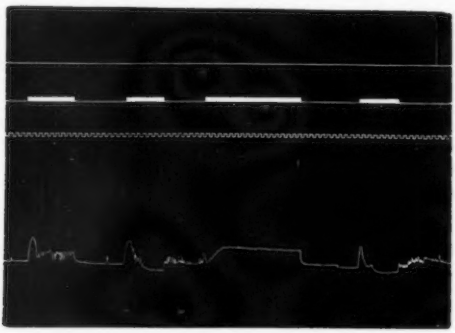

FIG. 13.

The typical "cord" effect is observed in the middle of the three curves as a prolonged tetanus after the clonic stage.

the cortex or corona radiata it is of course necessary to employ an excitation evoking a tetanus; I have done this with the mechanical method, by taking a tracing of a simple reflex action and a crossed reflex.

The simple reflex was obtained from the rectus femoris muscle through the right anterior crural nerve by excitation of the central end of the right sciatic, and the crossed reflex by the excitation of the central end of the left sciatic, the spinal cord being divided just above the middle of the dorsal region. In some cases a preliminary excitation of the cortex

[1] *Loc. cit.*

was first performed, but in others there was no interference with the parts above the section of the cord. The general results, *i.e.*, the work done by the muscle under these circumstances by simple and cross reflex action are as follows:—

We will first deal with the way in which the spinal cord responds to the simple reflex stimulus in order that we may define what is a characteristic spinal cord effect.

When the cord is excitable there is a very rapid response to the excitation, the weight is raised to the maximal height

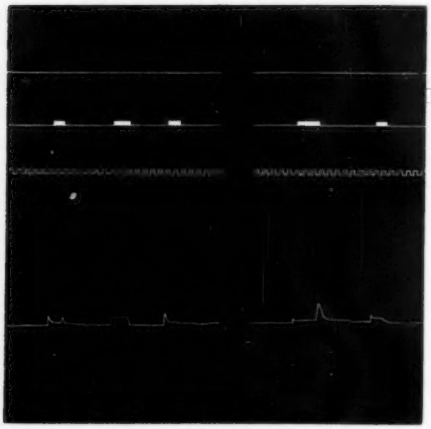

FIG. 14.

Simple reflex contractions showing double spike and large terminal spike.

almost at once, and not infrequently there is an initial spike on the curve which may be on the ascending portion of the curve, or even when the curve has reached its maximum. This initial spike I propose to call the "initial effect" to distinguish it from a terminal spike to be described below (see fig. 14).

It is not, however, necessarily of the same nature as the terminal spike, for apart from a genuine rapid contraction an initial spike may be drawn by the momentum of the apparatus. This, however, is easily distinguished from a real contraction rise. The line of the curve then

runs horizontally forwards, more or less, and with indications of a rhythmical or clonic series of spasms (cf. Schafer & Horsley, *loc. cit.*), and with a more or less rapid descent, owing to "fatigue," in accordance with what has to be described directly, until we arrive at the end of the excitation.

In 1884 I had noticed frequently in the case of a spinal cord excitation, the occurrence of a spike at the end of the curve when the excitation was shut off, such spike resembling the large single clonic spasms often observed at the end of a cortical discharge. In 1881 Sanderson and Gotch noticed that when the spinal cord of a skate is caused to to discharge the electrical organ of that fish, it does so with an initial spike and a terminal spike. This form of discharge of the spinal centre must be, therefore, something of a very fundamental character if we find it ranging through the animal kingdom at such wide intervals as these; and here, again, it seemed to me that one might be able to bring the phenomenon into prominent relief by exaggerating the excitability of the cord and producing a premature dissolution of the same.

This phenomenon, which we may call the "end effect" of the spinal cord discharge, is closely associated with the question of the existence of a true spinal "after effect," comparable to the clonic stage or after effect derived from the cortex when the stimulus applied to the latter ceases. Without trenching on what has been said, I have now to point out that although in a more normal condition of the spinal cord, that is to say, during the early stages of an experiment, there is often no after effect to be obtained from the spinal cord resembling the clonic stage of the cortical discharge; still, an after effect may be produced at any time.

The after effect of the spinal cord, however, is so entirely different to the clonic stage of the cortical discharge that it is quite unmistakable, and when it is associated with the ordinary tetanic first stage derivable from the spinal cord, it is then so sharply defined as to justify the expression I used above of "typical spinal cord effect."

The form of the after effect varies with the intensity of the stimulus, as shown in the accompanying tracings.

A weak stimulus of 500 from the Kronecker coil gives a spike-formed after effect, which in many cases can be found to be a delayed end effect. I have found every transition stage between these. The usual spiked end effect simply requires to be delayed one or two seconds to be then an after effect. This is the first form of spinal clonic stage or after effect, and may be compared with the end clonic spasm of cortical discharge.

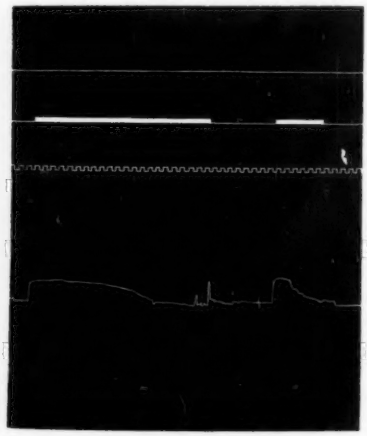

FIG. 15.

Excitation (reflex) of the spinal cord, showing difference of after effect, with (*a*) weak and (*b*) strong stimulus.

The second form is seen when a stronger stimulus (4,000 Kronecker coil) is used and when, as also will be seen directly, "fatigue" is showing itself in the nerve centres and assisting in increasing the delay, *i.e.*, intervals from two to six seconds. It, the after effect, begins slowly to reach a very moderate height and dies away slowly, sometimes exhibiting indications of a rhythm. (See fig. 15.) By taking a stimulus intermediate, say 1,000, one can obtain

an after effect, the character of which is intermediate between the two just described. It is interesting to note

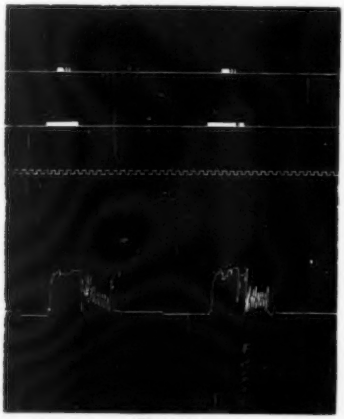

FIG. 16.

Cortex excitatory after effects to compare with the foregoing.

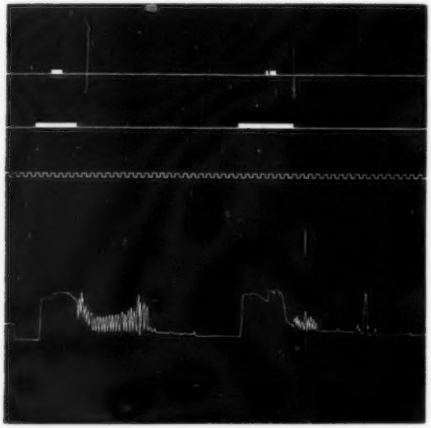

FIG. 17.

Cortex excitatory after effect; (*a*) common form; (*b*) delayed end effect.

that the difference in character of the after effects is not affected by exaggerating the excitability of the cord.

No doubt with different conditions of dissolution of the cord, varieties more or less clonic in character could be obtained from the spinal cord if the exact moment was fortunately hit upon; a very exceptional case, for instance, was obtained by Schafer and myself in 1884 from the spinal cord of a rabbit. (*Vide Journal of Physiology.*)

We may therefore conclude that *any* nerve centres, whether in the cortex or spinal cord is capable of discharging (as would have been said some time ago "spontaneously") after it has been thrown into excitation by some definite

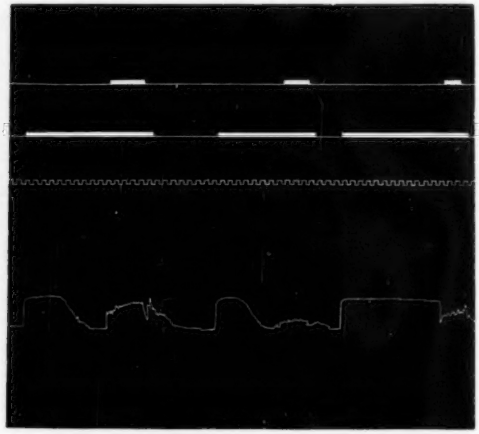

FIG. 18.

After effects varied by additional excitation of the spinal cord.

stimulus, but that this after effect of the activity of the centre varies both in *character* and *force* according to the nerve centre producing it.

I now wish to show example in fig. 19 that a capacity for producing a second output of work as it were, can be demonstrated to occur during the discharge of the spinal nerve centre itself and is in effect one variety of Franck's *tetanos a renforcement*, as observed by that author to result from excitation of the cortex, this is very well exhibited in

the dissolution of nerve centres by so-called fatigue and other depressing conditions, which subject I will now take up.

Effect of Disease State on the Output of a Nerve Centre.

Gotch and myself found[1] that the electrical effects obtained from nerve centres when the animal was suffering from some general pathological state, *e.g.*, from the severe disease known as distemper, were small. The same thing is true of course as regards the work done by the muscle

FIG. 19.

when the cortex is excited, and the character of the fatigue effect is that in accordance with the facts previously described the tetanus derivable from the cortex is very slowly developed soon fades down again to the abscissa line and that there is no tetanus in the clonic stage, the clonic spasms in fact simply start from the abscissa line and fall back thereto each time.

Another variety of the same thing in degree of discharge of the nerve centre under a hampering condition of this

[1] *Loc. cit. supra.*

kind is that instead of a clonically marked tetanus the cortical discharge consists of a series of closely aggregated clonic spasms.

As regards the spinal nerve centre discharge when affected by general depression effect, *e.g.*, disease, shock, etc., it is characterised by (1) great diminution of the work done; (2) slow development of the same and rapid failure.

Fatigue by Excitation and its Effect on a Nerve Centre.

Concerning the behaviour however of a normal nerve centre the most interesting phenomena are seen when it

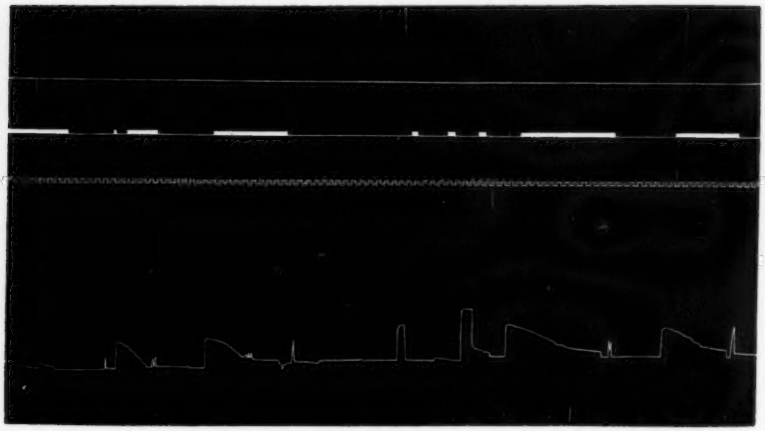

FIG. 20.

Fatigue effects on spinal nerve centres with strong stimuli. Note the spinal after effects.

appears to be exhausted during the continual application of an electrical stimulus. It might be expected perhaps that by Pflüger's laws the increase of the excitatory stimulus would be followed by an increase in the work done by the nerve centre, but this is not the case, and the whole subject it will be found, is a complex and interesting one.

Genuine Fatigue.

The easiest example is to take a reflex action. The following series of tracings show that with a stimulus of only 500 the degree of work done is very great. Prolonged excitation evoked continuous elevation of the weight, which

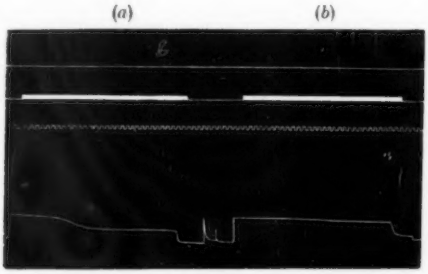

FIG. 21.

Comparison between fatigue of (*a*) reflex spinal centre; (*b*) fatigue of the muscle (on excitation of motor nerve).

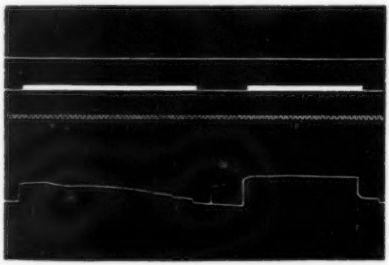

FIG. 22.

Fatigue of spinal centre compared with muscle; prolonged excitation.

is only very slowly and gradually lowered, whereas with an excitation of 4,000 the weight is maintained at its maximal height only for a short period and the descent to the abscissa line is very rapid.

I think it is questionable whether this is fatigue at all, or whether it is not rather an interference phenomenon, because if you throw into the cord an excitation of the

afferent end of the opposite sciatic nerve during the fall of the weight towards the abscissa (the original excitation of the same sciatic nerve being continued all the while), it is found that there is a rapid and powerful raising of the weight and also a marked after effect of the kind characteristic of the spinal cord discharge when excited by a strong stimulus.

It cannot be said therefore that the spinal centre is exhausted in the ordinary sense of the word, but that the effect observed is more allied to the process of "inhibition."

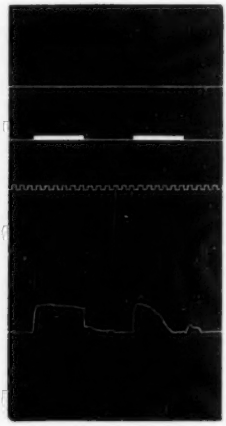

FIG. 23.

Effect of weak and strong excitation respectively on reflex centre.

To analyse this correctly, however, involves a review of our present knowledge of the arrangement of the antagonistic muscles in nerve centres. Attention has for some time been drawn by Sherrington, Beevor and others to the existence or non-existence of synergic action of antagonistic muscles, and has resulted in showing that when there is an active contraction of a flexor muscle, for example, that there is simultaneously an active relaxation of the extensor muscle.

This was first demonstrated by Sir Charles Bell and has subsequently been the subject of several papers. Several years ago Lowenthal discovered that excitation of the cerebellum evoked the same remarkable phenomenon, and the result of a long series of experiments by him and myself showed that this was due to the action of the cerebellum on the bulbospinal centres. In the same research we showed that the spinal centres themselves are arranged in an alternating fashion and that, for example, excitation

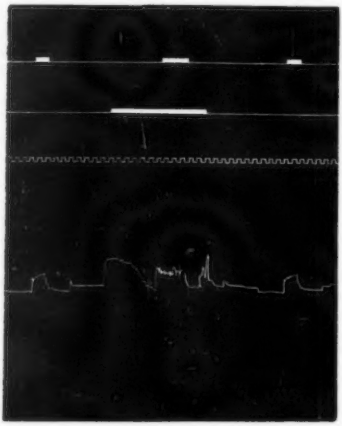

FIG. 24.

Combined excitation. The uppermost line indicates excitation of the central end of the opposite sciatic nerve. Powerful after effect.

of the central ends of similar nerves of the two limbs was followed by opposite motor effects, excitation of the central end of a nerve of the left side evoked a reflex contraction of a flexor, excitation of the same nerve on the opposite side produced a relaxation of that same flexor and contraction of the extensor.

Quite recently these observations have been amplified by Sherrington and Hering, and extended to an analysis of excitation of parts of the cortex cerebri. It is perfectly

clear, therefore, that every kind of nerve centre at present capable of being isolated must be assumed to have the two opposite functions of antagonistic muscles represented in it, the further conclusion from which is that whereas one function may predominate over the other it is possible that an unequal excitation might produce a *confused* result, and might indeed go further, might convert a former contraction of the muscle into an active relaxation of the same. This latter would be called by some inhibition and by others

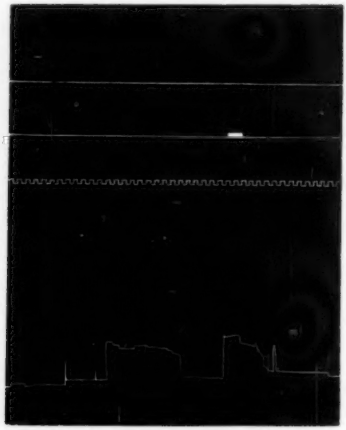

FIG. 25.

Comparison of after effect following strong excitation with absence of same after weak.

might be translated as being due to exhaustion and premature fatigue of the muscle.

It was for the purpose of distinguishing between these two possible conditions that I have employed the combination of reflex excitation with the primary excitation, the results of which have certainly taken us far beyond what Pflüger's original laws would seem to indicate; and yet at the same time, as the following tracings show, there is, of course, much truth in their general idea that it is

possible to raise the excitability of the spinal cord and get more work out of it. I can now show that the reciprocal innervation may be at the time being adverse, an example of this is shown in the case of a dog, where I found an unusual condition of things existing, namely, that excitation of the central end of the sciatic developed not contraction of the rectus femoris on the same side, but relaxation, just as though the hamstring muscle nerve had alone been stimulated (Sherrington), whereas excitation of the opposite sciatic nerve produced a small but well marked contraction

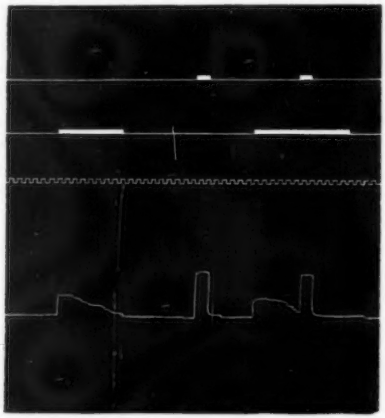

FIG. 26.

Excitation of motor nerve to muscle to show non-fatigue of muscle in contrast to the fatigue of the nerve centre.

of the muscle. This last effect, the crossed reflex, could be very notably increased by combining excitation of both nerves at once, showing that the general activity of the centre could be raised even under the adverse circumstances before referred to.

Using the same considerations to analyse so-called failure of the cortex on excitation, the following tracings obtained from the cortex of a perfectly normal animal during a series of observations by Dr. Levy on the fatigue

of the cortex cerebri, and for the loan of which I am indebted to him, show, I consider, the same state of affairs to be taking place, for the reason that the after effects are large and followed by the after excitation of the spinal cord to which I have before referred.

Herewith I also wish to record the occurrence of a very interesting delay of the onset of the clonic or after stage following excitation of the cortex cerebri, and which I have noted in cases where the nerve centres have been thrown

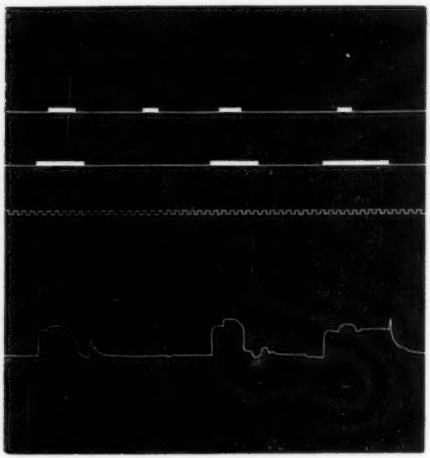

FIG. 27.

Excitation of both sciatic nerves (central end).

into an abnormal condition by excitation of the central end of the sciatic nerve. See in fig. 28 the interval especially in the second example between the tonic and clonic stages. Such delay is explicable upon the ground that the opposite reciprocal innervation has been heightened by the abnormal stimulation of the centre.

Correlative Phenomena Observed.

It will be remembered from what has been said above as regards the method of these experiments that the muscle

employed has commonly been the rectus femoris, biceps and supinator longus, but most especially the first of these. During the progress of experiments I noted that the tail muscles exhibited in a very striking way exactly the same degree and kind of contraction as the rectus femoris muscle,

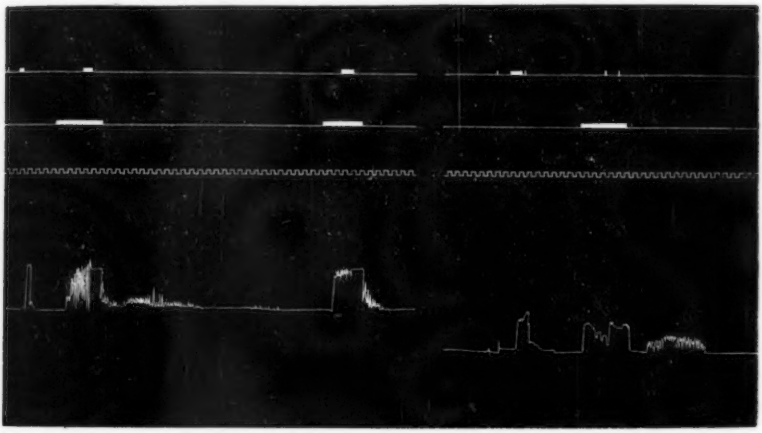

FIG. 28.

but any tonic, *i.e.*, tetanic effect, was more prolonged in the tail muscles than in the rectus. So, too, occasionally, the afferent excitation of the sciatic caused rhythmical contraction of the rectum. Schiff long ago drew attention to the excitability of the centre in the spinal cord for the representation of tail movements.

CASE OF EPILEPSY WITH TASTING MOVEMENTS AND "DREAMY STATE" — VERY SMALL PATCH OF SOFTENING IN THE LEFT UNCINATE GYRUS.

BY J. HUGHLINGS JACKSON, M.D., F.R.C.P., LL.D., F.R.S.,
AND
WALTER S. COLMAN, M.D., F.R.C.P.

PART I.[1]

IN this article are given further particulars of a case of epilepsy which I published[2] about ten years ago. I will call the patient Z. There was in this patient's slight attacks the "dreamy state" (commonly called "intellectual aura"). With at least some of his seizures there were certain movements of the mouth and tongue, tasting movements; these were, I suppose, the indirect "reflex" results of an epileptic discharge beginning in gustatory elements of a certain region of the cerebral cortex—(Taste region of Ferrier.) After Z's slight attacks, or after some of them, there were very elaborate actions during "unconsciousness." The patient died, January, 1894, from an overdose of chloral. Dr. Walter Colman found at the necropsy a very small lesion of the left uncinate gyrus. A report of Dr. Colman's examination of the brain will be found in the second part of this article.

History of the Case of Z.

I first saw the patient, a medical man, whom I call Z, in December, 1877. He was then 26 years of age. He

[1] This part is written by J. H. J.

[2] BRAIN, July, 1888. "On a Particular Variety of Epilepsy (Intellectual Aura); one Case with Symptoms of Organic Brain Disease." Case V. of that paper, p. 200, *et seq.* is the case of Z.

began to have slight epileptic attacks in 1871; they were so slight at first that he, to use his own words, "regarded the matter playfully and as of no practical importance." In 1874 he had a *haut mal*, and then for the first time knew the evil meaning of the slight seizures he had disregarded. He afterwards wrote out for me an account of his case, which, with his consent, I published (BRAIN, Part XLII., July, 1888, p. 201 *et seq.*). I may remark at once that there were no such signs of local intra-cranial gross organic disease (such disease as tumour, I mean) as optic neuritis and severe headache in any part of the patient's illness. I now give some details I obtained from Z in 1877.

He had that variety of the dreamy state I call "reminiscence"; he called it "recollection." In his slight attacks there was, he told me, a sentence in his mind which was as if well remembered. For example, if anyone was at the time speaking to him it would be as if he (Z) were trying to remember it, as if it were familiar, but yet he could not remember it. Again, he said—I give the words I hastily wrote in my case book, here intercalating other words in square brackets—"Attending to what was going on in [my] mind because [it was] interesting, and dim to what [was] going on outside." He could not, on recovery, remember what the "interesting matter" was. (*See* BRAIN, July, 1888, pp. 202-3, for further particulars of his dreamy state.) According to a report Z gave me in 1890, the reminiscence had disappeared, and from this report I gathered that he had had another kind of dreamy state with the reminiscence, and that this other kind occurred in his later fits. After the severe fit in 1874, in which he bit his tongue, he was subject both to slight and to severe attacks.

There was no evidence of any crude sensation warning of smell, taste, hearing or sight, nor did I ever afterwards hear of any; there was no epigastric sensation. There was no vertigo. Although there was no crude sensation warning of taste at any time, there were certain movements which, I think, imply an epileptic discharge of gustatory elements of the cortex cerebri. I did not, when I first saw the patient, obtain any information as to such movements.

He wrote of them (BRAIN, *op. cit.*, p. 204) as being, he had been told, "*somewhat like a modified and indistinct smacking of the tongue like a tasting movement, and is generally accompanied by a motion of the lower jaw* (italics in original), and sometimes by some twitching of the muscles round one or both corners of the mouth or of the cheeks, *but by no sense of taste in my recollection.*" (My italics.)

I ought here to say that several observers, one of them a highly accomplished medical man, who saw many of Z's attacks, never noticed any such movements as I have mentioned. They were very slight; in an attack (*vide infra*) which I witnessed, the noise made was only just audible. That they occurred in at least some of Z's slight attacks I do not doubt, because of the observation I made, and because he reported (BRAIN, *loc. cit.*, p. 204) that some others had noticed them. It may be that the patient had, during the period this medical man knew him, ceased to have such movements. Z wrote to me, May, 1890, a report in which, among other things, he says, comparing with previous years: "Although I have often asked observers to describe such a sound as I have made, and which in former notes [those published, BRAIN, July, 1888, p. 204] I described as a smacking of the tongue like a tasting movement, I have not found it so described, but rather as some indistinct attempt to speak."

He had a slight feeling of dread. It was not a fear of the fit; the dread came first and then the fit, or rather the rest of the fit.

He said the fit seemed somewhat within his control; he made a rule when it set in to try to do something definite, for instance, to speak. He gave, as another example, that he once made an excuse to pick up a ball. In August, 1880, he was still subject to slight fits. When a clinical clerk, he would have them in the wards of the hospital, but they were so slight that no one noticed anything particularly wrong. One medical man observed something peculiar, and so did several friends. Z was told that he appeared anxious to answer, but seemed to have lost the drift of the conversation—used in the connection some wrong word, and

yet appeared to search about for another word. This is not very definite, but serves to show how slight the slight attacks were at that time.

In the remainder of this article, I shall speak only of Z's slight seizures. I witnessed but two of them, and these I saw years after his first visit to me. In one he stopped talking to me, remained standing, and made slight, very slight, just audible (*vide supra*) smacking movements of his lips. I have no further details of this seizure; it occurred at the meeting of a Medical Society, and I had no opportunity of watching him throughout the attack. I knew that he was quite safe; no one but myself noticed anything amiss with him. On another occasion he was sitting in a room consulting me; he stopped talking—I have no remembrance of any smacking movements of his mouth on this occasion—his head was bent forward, but in a second or two, the paroxysm being then, I suppose, over, he looked up, and next (post-paroxysmal stage of actions) he leaned over one arm of his chair and felt about on the floor as if searching for something; next he did the like on the other side. Shortly, having a pin in his hand (how he got it I know not, perhaps from the floor) he made a feint of pricking my hand; the action was as if in fun, for he stopped well short of my hand and was smiling. This little affair was exactly after the manner of joking with a child, as if one said playfully to a child, "Now I'm going to prick you," and smilingly pretended an attempt to do so. Z, however, did not accompany his playful-seeming feint by words. Soon, perhaps a minute, afterwards, his actions, or I should say the irrelevant-seeming actions, ceased; he replied correctly to simple questions, and told me that it was not necessary for me to go home with him. He, however, looked confused and seemed strange. When we got to his house a few yards away, I thought he was fully recovered, and, as I was thinking of making another room on the ground floor of my house, I took the opportunity of speaking to him about a third room there was on the ground floor of his house. Among other things he said he used to breakfast there. I was surprised when he afterwards, next day, told me that he

remembered nothing from the time of being in my room consulting me (before the fit) to a little time after I left him at his own house.

These post-paroxysmal actions during what we clinically call unconsciousness were as elaborate and purposive-seeming as any of those of his normal self; such post-paroxysmal actions are very important in a medico-legal point of view. On another occasion there were post-epileptic actions by Z during "unconsciousness," of a kind which in a man fully himself would be criminal, and must have led to very serious consequences had not, fortunately, his condition been known. What he did was overlooked by those concerned.

In Z's report of his case (BRAIN, July, 1888, p. 206, paragraph 4), he speaks of examining and prescribing, when temporarily unconscious after an attack, for a patient. I now give a specimen of what he wrote—notes of a patient's case—in such circumstances. The words I have put in curved brackets were crossed out by Z; those in square brackets are my words. "For six or seven (years) weeks he has felt very discomfort of the throat and also for the swelling of the throat and (legs) arms and ——" [The word for which I have put a mark signifying a blank I cannot decipher]. Here is another specimen: "For the last few days his beginning (starting to walk ?) is more difficult for his tenderness of speechlessness and quick power of talk light swolleness of feet last three days." The words "starting to walk" in the square brackets were inserted later. Here is another specimen with Z's remarks on it: "I had written the above [*i.e.*, he had filled up the form for name, age, &c., of the patient] correctly and ascertained that the man had had gradually increasing œdema of the legs lately, when a *petit mal* came on with no overpowering impression, or indeed any distinct impression that I can recollect. I wrote the words below [below the heading] slowly and with difficulty, wishing to be correct; on reading them over they seemed to me correct; but I could not go on; I turned over the page and tried to describe the man, stared at him and felt too undecided to

write more than 'A rather';—could easily copy what I saw before me written down over the man's bed already as to the urine. Altogether it took two or three minutes. I remained undecided and disinclined to write, but able to talk for five minutes more." This is what Z wrote on this occasion: "For about the last fortnight about the legs are about the gradual for several debts of the" (Z adds, "no connection in thought traceable for the word 'debts'").

Still one more specimen of Z's writing. He wrote the following in a state, which at the time, he thought was full consciousness, but afterwards knew to have been defective consciousness: "There [then a word obliterated] was constant repetition of sickness for the last twenty-four hours. Abdomen [this word crossed out]. The sick [a word like 'neck,' joined to sick, crossed out] on the grateful rightnessness has felt a large knowfulness." Z wrote down when well what he thought this should have been: "The sickness has left a painful feeling on the right side; she has felt much fulness there."

The following specimen of Z's writing (it was written as notes of a case) was not corrected: "For the last 18 mos [mos, no doubt, is for months] years there has been some decided indefinite on R. side in dress circle." Moreover in the heading of the case in the part for occupation he made a mistake—"Hairdresship" for, I presume, "Hairdresser." The patient was a woman, but this may have been for her husband's occupation.

For localisation of the lesion I relied entirely on Ferrier's researches. I say this not only because credit should be given to the right man, but also because, having said what I have said, I can without immodesty put some facts more strongly than I otherwise should like to do. Let the following four statements be considered.

(1) Ferrier found that certain movements of the lips, tongue and cheek-pouches follow on artificial excitation of a certain region of a monkey's cortex. From the nature of these movements he inferred that that region contains gustatory nervous elements—that the movements were "reflex" consequences of the artificial excitation of those

elements. (2) I suppose that similar, or analogous, movements in human beings subject to certain epileptic fits, as in the case of Z, at the onset of their seizure, are "reflex" effects of epileptic discharges beginning in some gustatory elements of that cortical region which, according to Ferrier, is for taste. (3) I begged Dr. Colman to call on me before he went to make the necropsy on Z, in order to ask him to search the taste region of Ferrier on each half of the brain very carefully. (4) Dr. Colman (see Part II.) found a very small focus of softening in that region (in the uncinate gyrus) of the left half of the brain.

There was no trustworthy evidence to point to the half of the brain affected. Z wrote (BRAIN, July, 1888, p. 204): "I have no clear evidence that one side of the face is affected more than the other, and no clear evidence against it; from what little I can learn, if it is at all unilateral it is more on the right side than on the left; but the evidence is very scanty. I never notice it myself. I also never notice myself, but learn from others, that sometimes, especially if sitting, I give one or two light stamps on the floor with one foot, and in the only cases where this has been accurately observed it has been with the right foot."

There are many points for comment in Z's case. I refer to "Lectures on Epilepsy," *Medical Times and Gazette*, vol. i., 1879; BRAIN, July, 1880; BRAIN, July, 1888, for remarks on the group of cases to which this case belongs. In those papers I consider the Dreamy State in its association with crude sensations of smell and taste, and with the epigastric sensation and with chewing and tasting movements. In BRAIN, October, 1889, Dr. Beevor and I record a case of epileptic attacks with a crude sensation of smell and a dreamy state; there was tumour of the right temporo-sphenoidal lobe. In that paper Dr. Beevor refers to cases previously recorded of epileptic attacks with warning of smell. In the *Lancet*, January, 14, 1899, I published a note on what I call the Uncinate Group of epileptic fits. I there consider the asphyxia in slight fits of some cases of this group, and suggest, having regard to some researches of Mr. W. G. Spencer (*Transactions of Royal Society*, vol. clxxxv., 1894,

B, pp. 609 to 657), that the asphyxia may be owing to inhibition of the respiratory (medulla) centre by discharge spreading from a discharge lesion of the uncinate gyrus to the cortical centre for arrest of respiration; this arrest centre is close in front of the uncinate gyrus.

Part II.[1]

Pathological Report.

The autopsy was made thirty hours after death. The weather was very warm and damp. *Post-mortem* lividity was extreme, and decomposition was rather advanced.

Examination of Head.—The scalp was unusually thick, and firmly bound down to the periosteum. The *skull cap* was natural except for two symmetrical, flat, rounded elevations about half-an inch in diameter situated on the outer table, on each side of the bregma. The *dura mater* was unusually adherent to both parietal bones, but not elsewhere. The surface of the pia-arachnoid was normal, except beneath the bony prominences at the bregma, where there was some adhesion. Pacchionian bodies were large and numerous in this region, but inconspicuous elsewhere. There was no adhesion between the pia mater and the surface of the convolutions at any part.

Both hemispheres were fully convoluted, especially in the frontal and occipital regions, the convolutions following the usual arrangement. Their surface was perfectly normal in appearance, and the consistence of the brain was natural except in the left uncinate gyrus, where it could be felt that there was a patch of softening beneath the surface. The position of the softened patch is shown in fig. 1, which is drawn from a tracing of the brain after hardening in spirit. On a section being made through the patch it was found to be a small cavity, collapsed and almost empty, with indefinite walls, situated in the uncinate gyrus, $\frac{3}{8}$ inch below the surface just in front of the recurved tip of the uncus (*see* figs. 1 and 2). The existence of the patch

[1] This part is written by W. S. C.

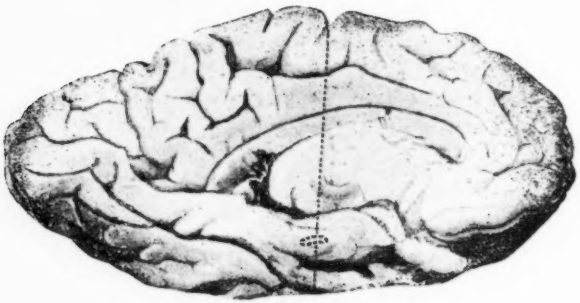

FIG. 1.

Drawing of internal surface of left hemisphere (case of Z). The black ring corresponds with the position of the cavity found, which is shown in section in Fig. 2.

FIG. 2.

Vertical section of left hemisphere (case of Z, from a tracing). Site of small cavity apparently due to softening, shown by black ring.

and its position were verified in the fresh specimen by Dr. Hughlings Jackson, Dr. Dawtrey Drewitt, Dr. James Taylor, and Dr. Guy Wood. The cavity was like those seen long after softening from thrombosis or embolism; there was no surrounding inflammation nor any indication that it was of recent origin. Search was made for an occluded artery in the neighbourhood, but without success. The other vessels of the brain did not show any signs of atheroma to the naked eye.

Unfortunately the uncinate region became so soft and friable during the hardening process that it was impossible to make satisfactory sections, and the microscope did not throw any further light on the cause of the lesion.

The urine contained a small amount of albumen, and there were early interstitial changes in the cortex of the kidneys.

There were many pulmonary infarcts apparently produced during the prolonged coma which was present for twenty-four hours before death, and the lungs themselves were extremely œdematous.

With these exceptions the organs were healthy.

A localisation of the lesion in this situation had been suggested by the presence of the "dreamy state" or "intellectual aura." In a paper read before the Medical Society of London in 1889 and afterwards published in BRAIN, Drs. Hughlings Jackson and Beevor published the case of a woman, 53 years of age, who had a tumour, the size of an orange, at the anterior extremity of the temporal lobe. During life she had numerous fits, with an intellectual aura and with "a horrid smell." A very instructive case recorded by Sander[1] is quoted in the paper by these authors. The patient had fits which were preceded by the warning of a "dreadful disagreeable smell," and he then had "*chewing movements of the jaws, and spitting of saliva.*" Later on he had convulsions about the face, but not in the limbs. He became very dull

[1] Sander, "Epileptische Anfälle mit subjectiven Geruchs Empfindungen bei Zerstörung des linken Tractus olfactorius durch einen Tumor." *Archiv f. Psych.*, vol. iv., p. 234.

mentally, so that few subsequent observations on his sense of smell or of taste could be made. On *post-mortem* examination a gliomatous tumour the size of half a large apple was found on the under surface of the brain, at the junction of the left frontal and temporo-sphenoidal lobes. Half the growth involved the anterior part of the temporo-sphenoidal lobe, the other part reached across the fissure of Sylvius involving the lower convolutions of the frontal lobe. Inwards the growth reached the middle line.

Reviews.

Die Bedeutung der Augenstörungen fur die Diagnose der Hirn- und Ruckenmarks-Krankheiten. Von Dr. Otto Schwarz, Privatdocent an der Universität, Leipzig. ("The meaning of disturbances of the eyes in the diagnosis of disease of the brain and spinal cord.") 1 vol., 8vo, pp. 100. Karger, Berlin. 1898.

For the past few years the interest in the connection of ophthalmology and general medicine has been steadily increasing, and has found its expression in the appearance of several works. Amongst others, the valuable book of Berger ("Les maladies des yeux dans leurs Rapports avec la Pathologie Générale,") and that of Knies ("Die Beziehungen des Sehorgans und seiner Erkrankungen des Körpers und seiner Organe"), treat of the relationship between eye disturbances and diseases of the nervous system. In these, however, the eye conditions are not viewed from the standpoint of neurological diagnosis, and in many handbooks on neurology and general medicine, they do not hold the important position they deserve. Dr. Otto Schwarz, therefore, feels justified in presenting this work, dealing more exactly with the meaning of eye trouble in the diagnosis of disease of the brain and spinal cord. He claims a certain authority in so doing, having had peculiar advantages for systematic work in this direction for many years. His endeavour has been to give to the neurologist the clearest possible picture of the signification of eye disturbance, and to assist the ophthalmologist in forming a comprehensive opinion, so that he may be of assistance to his medical brethren in the diagnosis of nervous diseases.

This work is a valuable one, in that it is an honest, capable effort to bring into concise and simple order the items of a very difficult and, it must be confessed, little known subject. Years of careful and progressive observation must pass before much certainty in this remarkably abstruse and peculiarly elusive field can be obtained.

There is no doubt that the author has built up his book as thoroughly as is possible, and has taken his facts from the most reliable sources. It is in some places laboured, not easy of expression, and as disconnected as a dictionary. Possibly this is not to be avoided to a certain extent, owing to the great difficulty of obtaining sound and sufficient information in some of the more or less rare diseases of the nervous system.

About twenty-eight pages out of the hundred which make up the book are given to Hysteria, and are remarkably good and instructive. This undue proportion is explained by the author as being due to three reasons: (1) the great variety of the symptoms; (2) that no systematic work, from the diagnostic point of view on this disease, exists in the German language; and (3) that the writer has had long and rich experience of the malady. The author has not been able to resist the temptation of giving us a new definition of Hysteria, which may or may not add to our power and facility of appreciating this abnormality.

As a work of reference this book will be very valuable, and should be in the possession of every neurologist.

H. Work Dodd, F.R.C.S., &c.

The Nervous System and its Diseases. By Charles K. Mills. 1 vol., large 8vo, pp. 1056. Pentland. 1898.

This is the latest of the large text-books on neurology which have been given us from the American continent, and it is, without doubt, the most extensive which has appeared in the English language since the monumental work of Sir William Gowers.

The volume is complete in itself, in so far as completeness is possible in a rapidly progressive subject; a copious bibliography and general index putting the reader in possession of the latest publications bearing upon the anatomical, physiological and clinical relations of neurological work.

The first portion of the book treats of the anatomy, physiology and chemistry of the nervous system. Into this part has been incorporated the terminology and nomenclature introduced by Prof. Wilder, of Cornell. Whether this terminology is likely to receive that general recognition and adoption which the author hopes, is a point on which some doubt may be thrown. However

desirable general conformity in nomenclature may be, it will be long before such classical terms as "foramen of Monro" and "lateral ventricle" are known as "porta" and "paracoelia" respectively.

The second portion of the work is devoted to various general considerations, such as etiology, methods of investigating cases, the practical applications of electricity, and the general therapeutics of nervous disease; which chapter is from the practitioner's point of view one of the best in the volume. Here we have detailed the important subjects of balneology, massage, systematised movements, and the "rest cure." Several pages also are given over to the uses and applications of drugs in the treatment of nervous disease.

Four hundred pages are given to the consideration of the organic diseases of the brain and its membranes. It is impossible, in this short review, to follow the author through the mass of valuable material which he has brought together to throw light on this subject. Suffice it to say that there is no known condition which has not received ample consideration. More especially would we refer to cerebral localisation in all its bearings, the chapters on which appear to be the most complete with which we have met.

In the remainder of the volume the affections of the special sensory, and of the motor cranial, nerves are considered. The book would occupy in neurological literature a position of pre-eminence were this the only subject of study. They are the most detailed and comprehensive which we have come across in our reading. Were it not invidious to draw comparisons between chapters, all of which are valuable and pregnant with facts of interest, we would particularly refer to those upon the special senses as being worthy of the closest study.

The reading of this volume encourages the hope that the author will give us the other, which shall include the remaining diseases of the nervous system, insanity, and the medical jurisprudence of nervous and mental disease, which he has conditionally promised in his preface.

W. Aldren Turner.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

June 30.—Dr. C. E. Beevor opened a discussion on "The Accurate Localisation of Intra-cranial Tumours," and Drs. Byrom Bramwell, Mitchell Clarke, and Sharkey took part in the discussion.

At the Clinical Meeting of the Society, held at 11, Chandos Street, W., on Thursday, October 27, 1898, at 8.30 p.m., the following cases were shown :—

Dr. Ormerod and Dr. Batten.—Idiopathic muscular atrophy (Landouzy-Dejerine type) in mother and son.

Case I.—M. S., 39, ten years ago first noticed weakness of left arm; both arms became gradually affected; five years ago began to experience difficulty in going up-stairs. No alteration has been noticed in the face, but the patient states that when a child, her mother did not like to go into the room in which she slept as her eyes were not closed during sleep, and seemed always to be looking at her.

Family History.—Married; three boys; no miscarriage. First child described below. Second and third boys said to be healthy. No history of a similar affection can be obtained in her family or their children. She has five sisters and two brothers.

Present Condition.—Facial atrophy; weakness of orbicularis oris and palpebrarum; atrophy of trapezius and rhomboids; "winging" of scapulæ; weakness of anterior tibials and peronei of both legs; pes cavus. K. Js. active; no A.C.; plantar slight.

Case II.—E. S., 5 years; never able to close the eyes well and sleeps with them open; two years ago had his canaliculi slit at Moorfields in consequence of tears falling over his face, and on one Christmas previous to this he was presented with a trumpet which he was unable to blow until his mother held the corners of his mouth.

Present Condition.—Very marked weakness of frontalis and

orbicularis palpebrarum and oris; weak zygomatici; body thin; all movements capable of being performed, but some weakness of anterior tibials and peroneal muscles. K. Js. present, equal.

Dr. BEEVOR.—Unilateral wasting and paralysis of lower half of trapezius.

Patient is a girl, aged 20, who had typhoid fever and "brain fever" at 12 years, and nine months after her scapula was noticed to protrude. At present there are no fibres of the trapezius remaining below those which pass from the second dorsal spine to the inner end of scapular spine, and the rhomboid is visible. No other muscles affected.

The want of action of the muscle is seen in advancing or abducting the arm to the horizontal line, but beyond that point the serratus magnus acts.

The condition is probably a neuritis or a poliomyelitis.

Dr. H. SINEGAR.—Case of idiopathic muscular atrophy.

A female, aged 26, noticed weakness in her right arm eleven years ago, her attention being first attracted to it by difficulty in doing her hair. Wasting and weakness soon involved the left arm and steadily increased on both sides. She has been unable to put her hands on her head for five years.

Family History.—Negative.

Present Condition.—Weakness of orbicularis palpebrarum; winging of scapulæ; wasting of shoulder and arm muscles. Muscles affected; on right side, supinator longus, pectoralis major, serratus magnus and rhomboids practically absent, biceps and trapezius markedly wasted; on left side biceps, supinator longus, serratus magnus and trapezius practically absent, triceps and rhomboids greatly wasted. The deltoids and supra-spinati are distinctly hypertrophied on both sides. K. Js. active; no ankle clonus.

Dr. F. W. MOTT.—Arsenical neuritis from local action of the poison followed by general degenerative effects upon the nervous system.

A temperate man, aged 56, who had never had any illness before, was employed at chemical works to scrub the sheep-dipping compound from the indiarubber clothes of the men employed in its manufacture. This compound contains a large quantity of arsenic and alkali. About six weeks after he had been at the work he noticed a numbness and tingling in the fingers of the right hand, with gradual and progressive loss of power, so that he was obliged later on to give up the work because he could not hold the scrubbing-brush. He had con-

siderable loss of sensation to pressure, heat and cold over the right forearm and hand, and he was unable to use his hand. The reaction of the muscles to the faradic current was diminished. He had had no general symptoms of arsenical poisoning.

Six months later he came complaining of trouble in passing water, and although sensation in the arm and hand had improved, there was considerable loss of power, and the skin of the hand was smooth and glossy. To the local condition, however, there were added new symptoms, viz., paresis in the lower limbs with spastic gait and very exaggerated knee-jerks.

While the hand has somewhat improved so that he can feel a light touch, the paresis and spastic gait has increased, and he has, therefore, been admitted to Charing Cross Hospital.

Dr. ORMEROD.—Hereditary Chorea. A woman aged 50. Subject to constant irregular movements of the face, trunk, and limbs, which in character recall those of a mild chorea. No other physical abnormality. No marked defect of intelligence, but she appears to be rather forgetful and garrulous.

The movements began two and a half years ago, after a shock; she had been dismissed and forcibly ejected by her employer.

No previous chorea. No rheumatism.

Mother died of a similar complaint, aged 57, after fifteen years' illness. *Mother's mother said to have had the same* disease. No insanity known in her family.

Of eight brothers and sisters only one survives (a sister aged 52, healthy). Of the rest, two brothers died in early manhood, and the rest quite young. None had this complaint. Of six children, three died in infancy, *one aged* 20, *of rheumatic fever*, two survive and are in good health.

Mr. LAKE and Mr. BALLANCE.—Case of recovery from temporo-sphenoidal abscess.

Mr. D., aged 33. Long standing left otorrhœa with sudden onset of intra-cranial symptoms; namely, headache, vomiting, slight right hemiplegia, pupils dilated, sluggish, left optic neuritis, no hemianopia, partial aphasia, alexia, and agraphia, complete left facial palsy, copious purulent flow from left ear, pulse 44, temperature 98° F.

January 26, 1898. *Operation.*—Pus seen pouring through attic. ʒii. pus evacuated from temporal lobe. Drainage of abscess three weeks. Complete recovery.

Dr. BEEVOR and Mr. BALLANCE.—Case of removal of sub-cortical cerebral tumour.

Mrs. S., aged 39, admitted into National Hospital, May 21, 1894.

Symptoms.—Headache, vomiting, optic neuritis, aphasia, partial right hemiplegia, right hemi-anæsthesia of cortical type.

First operation, July, 1894. Large infiltrating mixed-celled sarcoma, removed from upper part of left parietal region.

Re-admitted February 22, 1897. Almost complete aphasia—more paralysis, headache, &c.

Second operation, March, 1897. 5½ ozs. of tumour removed.

July, 1898. Left hospital able to walk alone; no aphasia, &c.

Dr. TAYLOR and Mr. BALLANCE.—Case of Trigeminal Neuralgia.

Mrs. N., aged 66, ten years ago commenced to have attacks of pain in right side of face. Meckel's ganglion removed five years ago. Pain absent fourteen months; then suddenly recurred. Last three months violent exacerbations affecting all three divisions of right fifth. Admitted National Hospital, August 10, 1898. Urine contained trace of albumin. Operation (Krause's) in three stages, August 18, 22, and 26. Result: complete relief from pain; taste sensation on posterior part of right side of tongue abolished.

Dr. LUNN and Mr. BALLANCE.—Case of removal of cerebellar tumour, three years and eleven months after the operation. The case was shown before at a meeting of the Society eighteen months ago.

At the Pathological Meeting of the Society, held at 11, Chandos Street, Cavendish Square, W., on Thursday, December 15, 1898, at 8.30 p.m., the following communications were made:—

Dr. MOTT.—*Changes in the brain, muscles, and other organs, found in persons having died with prolonged epileptiform convulsions.* The brain and spinal cord were examined by the Nissl and the Weigert iron methods in the following cases:—

(1) Boy, who died probably from sunstroke; death preceded by 22 fits and a temperature of 108° F. (2) Thrombosis of the great anastomatic veins of Trollard, occurring first in the left hemisphere, then in the right. The patient had a succession of right-sided fits, then a succession of left-sided fits; he died comatose with a temperature of 107·5° F. (3) Three cases of *status epilepticus* occurring in young and previously physically healthy individuals. Death ensued from asphyxia and heart failure, with enormous venous engorgement of the organs. (4) Case of rupture of middle meningeal artery on the right side followed by over

400 left-sided fits; the patient was trephined but died shortly after. (5) Case of a middle-aged man, said to be an epileptic; died after seven fits. *Post-mortem* discovered advanced chronic Bright's disease. ? Convulsions uræmic. (6) Spinal cord of a case of general paralysis; temperature 109° F., death. *Post-mortem:* hæmorrhage from spinal veins, causing hæmorrhagic pachy-meningitis spinalis universalis. (7) Case of septicæmia with prolonged pyrexia, terminating in hæmorrhages and a very severe anæmia, so that the blood showed an enormous number of nucleated corpuscles, and yet the cortical pyramidal cells appeared normal.

In the cases 1 to 3 inclusive, a very similar condition of things was found; namely, venous congestion, stasis, and minute hæmorrhages of the brain, and under the pia mater. Œdema with distension of the perivascular and perineuronal lymph spaces; arterio-capillary anæmia and marked changes in the chromatic substance of the cortical pyramidal cells.

In some instances, owing to the distension of the lymphatic spaces, a continuation can be made out between the lymph space around a capillary and the lymph space in which a cell lies. It would thus appear that there is a canalicular system of lymphatics in the brain.

Owing to the distension of the lymph space, it is impossible to say whether the cell is apparently or really shrunken. The nucleus is usually enlarged, and the whole cell stained a uniform colour; the normal Nissl bodies cannot be seen either on the protoplasmic processes or in the body of the cell. If the section be stained with polychrome blue or methyl blue and safranin, then examined with a high power, it will be observed that the body of the cell consists of a fine net-work stained pink. Along these delicate pink lines, extremely fine, blue particles can be seen encrusted—the remains of the chromatic substance at the nodal points of the trabeculæ look coarser, owing to aggregation. The normal spindle-shaped chromatic bodies on the protoplasmic processes have either altogether disappeared, or are extremely attenuated. The only part of the cell which retains its normal blue stain is the nucleolus. These cells therefore present a marked contrast to the cortical pyramidal cells of Case 7. Examination of the anterior horn cells of the spinal cord, and of their homologues in the medulla was not made in Case 1, but in all Cases under 2 and 3 comparatively slight changes only were made out. These results agree strikingly with observations which I made of the cortical pyramidal cells of monkeys, in

which Dr. Leonard Hill had ligatured two carotids and one vertebral artery, and show that local rather than general conditions were the cause of the marked changes observed in the cortical pyramidal cells as compared with the spinal motor cells.

We will now consider the general causes which might occasion the changes. (1) Pyrexia: This might have been the cause in Case 1, because the cord was not examined; but in the other cases the marked changes described in the cortical pyramidal cells were not found in the spinal cells. The anterior horn cells of the general paralytic who died with a temperature of 109° F., certainly showed changes similar to those met with in the cortical cells of the other cases. This may have been due to the pyrexia, for it is unusual in general paralysis to find very marked changes in the chromatic substance of the anterior horn cells, and I have never before seen such universal affection. At the same time it must be remembered that in consequence of the venous hæmorrhage profound circulatory disturbances with venous congestion and stasis must have occurred in this case. (2) Exhaustion from discharge of energy in the production of the fits. If this were so, the anterior horn cells should be affected as much as the cortical cell; moreover, in the animals that died after ligature of vessels, the same changes were observed, whether they had fits prior to death or not. Again, in Case 4, I was unable to discover any appreciable difference in the appearance of the cells of the two hemispheres, although there were 400 left-sided fits. These facts point to local circulatory conditions being the main cause of the difference in the appearance presented by the cells of the cerebral cortex. Leonard Hill has shown that any increase in the venous blood in the closed cranial cavity is accompanied by a corresponding diminution of arterial blood, so that venous congestion, stasis and exudation, will cause arterio-capillary anæmia in the brain. But we have seen that the cells of the medulla do not suffer to anything like the same extent as the cells of the cortex. Consideration of the veins of the brain shows that the return of blood of nearly the whole external and the upper part of the internal surface of the hemisphere is by veins which run upwards to enter the longitudinal sinus. Backward pressure of venous blood would much more readily lead to stasis and congestion in these veins than elsewhere. Exudation would be more abundant, and carbonic acid tension in the regions which are drained by these veins, greater than elsewhere. Probably this increased CO_2 tension increases the excitability of the neurons.

It is suggested that the marked bio-chemical changes observed in the cortical pyramidal cells in the cases referred to, is due primarily to the alteration of the lymph environment. If anoxæmia does not of itself produce this change it is possible that increase of carbonic acid or other products in the lymph may be the principal cause; although it is not denied that insufficiency of oxygen and excessive activity may play important parts.

Changes in other tissues—such as striped muscle, heart muscle, liver and kidney. I have examined microscopically (staining by the Marchi method) these tissues in five fatal cases of status epilepticus, and in a large number of general paralytics who have died after prolonged epileptiform seizures, and have found that they are the seat of extensive early fatty degeneration; although to the naked eye, *post-mortem*, this condition would not usually be suspected. The cause of such marked fatty change in the muscles in cases of status epilepticus in persons who were a few days previously in good bodily health is difficult to explain. The fat must come from actual tissue waste with incomplete oxidation of the fat owing to venous condition of the blood. It is possible that fatigue products may play a part, for Araki, working in Hoppe-Seyler's laboratory has shown that lactic acid appears in the urine after prolonged epileptic convulsions.

Dr. F. E. Batten.—"Congenital abnormalities in the nervous systems of three children." (1) Non-development of left cerebral hemisphere (growth arrested about the 7th month of fœtal life), absence of left pyramid, absence of left fillet, diminution in size of right superior peduncle of cerebellum, left olive possibly a little smaller than right, cerebellum of normal size.

Inferior peduncles of cerebellum are of equal size, and posterior longitudinal bundles are normally developed. Below the decussation of the pyramids the cord appears almost normal, except that the right side is slightly smaller than the left.

The specimen was taken from a child aged 6 months, who had had fits since birth, and had congenital absence of the left thumb. The child had an obviously asymmetrical head, knee-jerks very active, equal; right arm-jerk greater than left. During a convulsion the eyes were turned to the right, the left leg drawn up, the left arm flexed, and the right leg rigidly extended.

(2) *Hydromyelia and congenital defect of the cerebellum* in a case of encephalocele of the occipital region from a child aged 4 weeks. The encephalocele was removed, and after death it was found that the cyst was in communication with the 4th ventricle.

The cerebellum was very defective, only the lower portion of the lateral lobes being normally developed. The middle lobe was absent. There was separation of the posterior columns extending through the cervical region, and from the 3rd to 9th dorsal segments, there was dilatation of the central canal, which was largest at the level of the 5th dorsal segment. The lumbar cord was normal.

(3) *Heterotopia* in the dorsal and cervical regions of the spinal cord, in association with spina bifida in a child aged $1\frac{4}{12}$.

Drs. H. H. Tooth and Risien Russell.—"Acute Myelitis with Bacteriological Examination." A man, aged 36 years, previously healthy with the exception of an attack of influenza four or five years before the illness which necessitated his admission into the National Hospital for the Paralysed and Epileptic, Queen Square. No direct or indirect history of syphilis or other venereal disease could be obtained. A month before death flaccid paraplegia of rapid onset, in which right foot at first escaped, ushered in by pain and tingling in the back, between the shoulders, radiating to front of abdomen and down inferior extremities, where there was also a subjective feeling of numbness. Temporary tingling and numbness in hands. Three weeks before this he felt out of sorts and languid, and experienced pain between shoulders radiating to front of abdomen. Movements of right arm and hand weaker than left, except at shoulder (patient right-handed). Diaphragm and intercostals normal. Left pupil larger than right, but they reacted normally. Slight comparative anæsthesia to light touches and pricks below level of third rib in front, and spine of scapula behind with inability to distinguish between heat and cold over blunted area, everything being called hot. Knee-jerks at first absent; subsequently elicited on reinforcement, though exceedingly feeble. Retention of urine, with overflow dribbling. Constipation. No bedsores, but blister on sole of right foot, result of a burn. Skin of dorsum of left toes glossy. Diminished reaction to faradism in muscles of lower extremities, and to galvanism, K.C.C. = A.C.C. in all muscles below knees except anterior tibial group on left side in which A.C.C. > K.C.C. Thigh muscles K.C.C. = A.C.C. and in some K.C.C. very slightly > A.C.C. Urine: a trace of albumin. Pyrexia—101° to 104° F. Developed severe diarrhœa, became collapsed and died.

At the autopsy cord of normal consistence throughout, but on section and subsequent microscopic examination marked evidences of disseminated myelitis with minute hæmorrhages detected. Three diplococci, closely similar morphologically, were cultivated

from the substance of the spinal cord. Two of these resembled each other somewhat in their mode of growth on nutrient media, while the characters of the growth in the case of the third organism were entirely different from those of the other two. Inoculation experiments were conducted on the monkey, cat, guinea pig, and mouse, but only one of the diplococci proved pathogenic, and this only to the guinea pig.

Rules

AND

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1898,

WITH THE

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REPORT OF COUNCIL.

JANUARY 13, 1898.

The Council has much pleasure in reporting the continued success and prosperity of the Society. The Members now number 187.

The Inaugural Address was delivered at the Annual General Meeting on January 14th, 1897, by Dr. G. H. SAVAGE, on "Heredity in the Neuroses."

A special feature in the year's programme has been the delivery of the first Hughlings Jackson Lecture on December 8th, 1897, by Dr. HUGHLINGS JACKSON himself, in whose honour the Lectureship was founded by the Society last year. The subject of the Lecture was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

A satisfactory arrangement has been made with Messrs. MACMILLAN & Co. in view of the increased cost of publishing *Brain*, as they have undertaken to hand over to the Society one-third of their share of the net profits from the sale of the Journal, as a contribution towards the cost of illustrations.

The Council regrets the loss sustained by the Society by the death of an illustrious Honorary Member, Professor Du Bois Reymond, of Berlin; also of Dr. JAMES CAGNEY and Dr. E. FRICKE.

The Proceedings have been published at greater length in *Brain* than in former years, and an abstract appended to this Report shows the character and value of the work of the Society during the past year.

Proceedings of the Society during the year 1897.

January 14th.—Dr. G. H. SAVAGE, Inaugural Address, "Heredity in the Neuroses," *Brain*, vol. xx., p. 1.

March 4th.—Dr. F. E. BATTEN made a communication on "The Muscle Spindle under Pathological Conditions," and illustrated his paper by lantern slides and by specimens under the microscope. Professor SHERRINGTON and Mr. VICTOR HORSLEY also made communications on the Muscle Spindle, and illustrated their remarks in the same way.

April 22nd.—Dr. FERRIER, "Two cases of Myoclonus Epilepticus." Dr. DONKIN (1) "Probably Subacute Poliomyelitis in a man aged 45 years"; (2) "Tabes Dorsalis with a great Preponderance of Sensory Disturbance"; (3) "Pseudo-hypertrophic Paralysis in an Advanced Stage." Dr. GOSSAGE, "Progressive Muscular Atrophy Commencing at the Early Age of 10½ years." Dr. LUNN, Dr. BEEVOR, and Mr. BALLANCE, "Case of Removal of a Cerebellar Tumour, two years and five months after the operation." Dr. JAMES TAYLOR, "Syringomyelia with Bulbar Symptoms." Dr. Harris, "Syringomyelia with Supposed Hæmorrhage into the Cavity."

June 3rd.—Professor SCHÄFER, "Is there a Direct Relationship between the Motor Paralysis produced by Lesions of the Cortex and Loss of Sensibility, Muscular or other?" The paper was illustrated by Lantern Slides, three living monkeys and the brain of a fourth that had been killed. Professor BOYCE and Dr. WARRINGTON, "Contributions to the Anatomy of some of the Ascending and Descending Nerve Tracts in the Fowl." (Lantern Demonstration.)

October 28th.—Dr. J. A. ORMEROD on behalf of Drs. SANGER BROWN and ADOLF MEYER, "Morbid Anatomy in a case of Hereditary Ataxy of the type described by Sanger Brown." (Illustrated by specimens under the microscope.) Drs. SAMUEL GEE and H. H. TOOTH, "Case of Hæmorrhage in the Region of the Pons Varolii, almost entirely on the Right Side." (Lantern Demonstration.) Dr. W. A. TURNER, "Symmetrical Softening of the Pyramids and Inter-olivary Strata." (Lantern Demonstration.) Dr. JAMES TAYLOR and Mr. C. A. BALLANCE, "Case of Tumour of the Right Lateral Lobe of the Cerebellum." Dr. RISIEN RUSSELL, "Degenerations in the Spinal Cord after Removal of a Large Tumour from one Cerebral Hemisphere, and in the case of a Tumour of the Lumbo-Sacral Cord."

December 8th.—Dr. HUGHLINGS JACKSON delivered the first Hughlings Jackson Lecture, the subject of which was "Remarks on the Relations of Different Divisions of the Central Nervous System to one another and to Parts of the Body."

BALANCE SHEET, 1897.

NEUROLOGICAL SOCIETY.

Receipts.	£	s.	d.	£	s.	d.
To Balance—						
General Balance ...	41	6	11			
Sir W. Broadbent ...	8	8	0			
				49	14	11
Subscriptions for current year				171	4	0
Do. in advance				1	0	0
Do. in arrears				12	0	0
Share of profit on "Brain"				46	10	4
Contribution by Messrs. Macmillan towards cost of illustrations in "Brain"				15	0	0
				£295	9	3

Expenditure.	£	s.	d.	£	s.	d.
By Stationery and Printing—						
Pulman & Sons	7	15	3			
Bale & Danielsson ...	9	9	9			
				17	5	0
Illustrations—						
Bale & Danielsson ...	75	11	6			
Pearson	2	0	0			
				77	11	6
Microscopes—Baker				6	16	0
Hire of Rooms and Refreshments—						
Royal Medico-Chirurgical Society				12	12	0
Medical Society	21	7	0			
Fielder (Univ. Coll.) ...	1	7	6			
				22	14	6
Messrs. Macmillan for publishing "Brain"				102	14	3
Petty Cash				0	18	0
Bank Charges				0	4	6
Indexing "Brain"				8	8	0
Balance				46	5	6
				£295	9	3

CHARLES E. BEEVOR, *Hon. Treasurer.*

Audited and found correct, December 17th, 1897. { J. B. LAWFORD. W. S. COLMAN.

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF LONDON.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Pound, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—Absence of any Member residing within the Metropolitan area from all Meetings held during the year shall be considered as equivalent to resignation.

16.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, and ten Councillors.

17.—Five Members of the Council shall form a quorum.

18.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

19.—The office of Vice-President shall be tenable for two years; one Vice-President being elected every year.

20.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

21.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

22.—One full week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

23.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

24. —There shall be six ordinary meetings annually, of which the first held in each year shall be the Annual General Meeting.

25.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

26.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty members.

27. —The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

28.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

29.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

30.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unless it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward.

Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by A. de Watteville, M.A., M.D., B.Sc.

HONORARY MEMBERS.

Mr. Herbert Spencer, London.
Professor Wundt, Leipzig.
„ Hitzig, Halle.

CORRESPONDING MEMBERS.

Professor Erb, Heidelberg.
„ Golgi, Pavia.
„ Goltz, Strasburg.
„ Grasset, Montpelier.
„ Jolly, Berlin.
„ Luciani, Rome.
„ Munk, Berlin.
„ Obersteiner, Vienna.
„ Retzius, Gustaf, Stockholm.
„ Ribot, Paris.
Dr. Weir Mitchell, Philadelphia,

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. Hughlings Jackson, M.D., F.R.S.
1887 Sir Samuel Wilks, Bart., M.D., F.R.S
1888 Sir James Crichton-Browne, M.D., F.R.S.
1889 Jonathan Hutchinson, F.R.S.
1890 Thomas Buzzard, M.D.
1891 John S. Bristowe, M.D., F.R.S.
1892 Henry C. Bastian, M.D., F.R.S.
1893 Edward A. Schäfer, F.R.S.
1894 David Ferrier, M.D., F.R.S.
1895 Sir William H. Broadbent, Bart., M.D., F.R.S
1896 Alexander Hill, M.A., M.D.
1897 George H. Savage, M.D.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

—:o:—

EXPLANATION OF ABBREVIATIONS.

O.M., Original Member. | Tr., Treasurer.
Pres., President. | Sec., Secretary.
V.P., Vice-President. | C., Member of Council.
H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected.

1892 ALEXANDER, ROBERT REID, M.D., The Asylum, Hanwell, W.

1897 ANDERSON, H. KERR, M.B., B.Sc., 9, Park Terrace, Cambridge.

1894 ANDRIEZEN, W. LLOYD, M.D., Darenth Asylum.

1891 BALLANCE, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897.)

O.M. BARLOW, THOMAS, M.D., F.R.C.P., 10, Wimpole Street, W.

,, BASTIAN, H. CHARLTON, M.D., F.R.C.P., F.R.S., 8A, Manchester Square, W. (Pres., 1892; V.P., 1891; C., 1886-9, 1893-5.)

1897 BATTEN, FREDERICK E., M.D., M.R.C.P., 124, Harley Street.

,, BEACH, FLETCHER, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey.

,, BEEVOR, C. E., M.D., F.R.C.P., 33, Harley St., W. (Tr., 1894; C. 1893.)

1888 BIRT, ERNEST, M.D., L.R.C.P., M.R.C.S., West Riding Asylum, Wakefield.

1898 BOLTON, JOSEPH SHAW, M.D., B.S., B.Sc., Mason College, Birmingham.

1896 BOND, C. HUBERT, M.D., B.Sc.Edin., London County Asylum, Banstead.

1892 BOWLBY, ANTHONY A., F.R.C.S., 24, Manchester Square, W.

1889 BRADFORD, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 60, Wimpole St., W.

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894.)

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 2, Henrietta Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., L.R.C.P., M.R.C.S., 18, Warwick Road, Maida Vale.

O.M. BROADBENT, Sir WM. H., Bart., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.P., 1893; C., 1886-7, 1895.)

1894 BRODIE, T. GREGOR, M.D., L.R.C.P., Lindfield, Uxbridge Road, Surbiton.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., 34, Washington Street, Chicago.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 13, Alva Street, Edinburgh. (C., 1895.)

O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

,, BRUNTON, T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896.)

1895 Bryan, Frederick, M.B.Dunelm, M.R.C.P., The Asylum, New Southgate, N.

1895 Bryant, John Henry, M.D.Lond., L.R.C.P., Guy's Hospital, S.E.

1890 Bury, Judson Sykes, M.D., M.R.C.P., 10, St. John Street, Manchester.

O.M. Buzzard, Thomas, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.P., 1888-9; C., 1886-7, 1891-5.)

1889 Carter, Robert Brudenell, F.R.C.S., 31, Harley Street, W.

1887 Cattell, J. McK., M.A., Ph.D., Columbia College, New York, U.S.A.

1891 Clarke, J. Michell, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol.

O.M. Cobbold, C. S. W., M.D., F.R.C.P.Ed., Bailbrook House, Bath.

1892 Cole, Robert Henry, M.D., M.R.C.P., M.R.C.S., 53, Upper Berkeley Street, Portman Square.

1890 Colman, W. S., M.D., F.R.C.P., 22, Wimpole Street, W.

„ Corner, Harry, M.D., L.R.C.P., Brook House, Southgate, Middlesex.

1894 Cuming, James, Prof., M.S., F.R.C.P.I., 33, Wellington Place, Belfast.

1895 Davidson, Andrew, M.B., C.M., The County Asylum, Dorchester, Dorset.

1897 Dawson, William R., M.D., B.Ch., Farnham House, Finglas, Co. Dublin, Ireland.

1894 Dean, H. Percy, M.D., F.R.C.S., 69, Harley Street, W.

O.M. Donkin, H. B., M.D., F.R.C.P., 108, Harley Street, W. (C., 1897.)

„ Dreschfeld, Julius, M.D., F.R.C.P., 325, Oxford Road, Manchester. (V.P., 1898.)

„ Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1889 Eccles, A. Simons, M.B., M.R.C.S., 23, Hertford Street, Mayfair, W.

O.M. Edmunds, Walter, M.B., F.R.C.S., 75, Lambeth Palace Road, S.E.

„ Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1894; V.P., 1892-3; C., 1886-90-95.)

„ Foster, Michael, M.D., F.R.S., Shelford, Cambridge.

1888 Fox, Arthur, M.B., F.R.C.P.Ed., 16, Gay Street, Bath.

O.M. Fox, E. Long, M.D., F.R.C.P., Church House, Clifton, Gloucestershire.

„ Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (C., 1896; V.P., 1897.)

„ Gee, Samuel, J., M.D., F.R.C.P., 31, Upper Brook Street, W.

„ Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1890 Goodall, E. W., M.D., Eastern Hospitals, Homerton, E.

1897 Gossage, Alfred Milne, M.B., M.R.C.P., 54, Upper Berkeley Street.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, Dundas, M.D., F.R.C.S., 8, Upper Wimpole Street, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W.

1895 Guthrie, Leonard, M.D., M.R.C.P., 15, Upper Berkeley Street, W.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., 9, Ridgemount Gardens, W.C.

O.M. HAMILTON, D. J., M.B., F.R.S.E., University, Aberdeen.

1897 HARRIS, WILFRED J., M.D., M.R.C.P., 7, Craven Mansions, Craven Terrace, W.

1891 HAWKINS, HERBERT PENNELL, M.D., F.R.C.P., 56, Portland Place, W.

O.M. HAYCRAFT, J. B., M.B., F.R.S.E., The University, Cardiff.

1891 HEAD, HENRY, M.D., C.M., M.R.C.P., 61, Wimpole Street, W.

O.M. HILL, ALEX., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.P., 1895; C., 1892-94.)

,, HOPKINS, JOHN, F.R.C.S., London Sick Asylum, Cleveland Street, W.

,, HORSLEY, VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.P., 1896; C., 1892-3.)

1889 HUGGARD, WILLIAM R., M.D., M.R.C.P., Davos Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.D., M.S.Ed., Montague Road, Felixstowe.

1889 HUMPHRY, LAURENCE, M.D., M.R.C.P., 3, Trinity Street, Cambridge.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., Bethlehem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887-92.)

,, JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 6, Manchester Square, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 9, Upper Wimpole Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

,, KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W.

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Trinity College, Cambridge. (C., 1898.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W.

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W.

,, LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., Claybury Asylum, Woodford, Essex.

1897 LOWENTHAL, MAX, M.D., M.R.C.P., 84, Princes' Road, Liverpool.

1891 MACDONALD, PETER WILLIAM, M.D., C.M., The County Asylum, Dorchester, Dorset.

,, MACKENZIE, HECTOR WILLIAM GAVIN, M.D., F.R.C.P., 59, Welbeck Street, W.

O.M. MACKENZIE, STEPHEN, M.D., F.R.C.P., 18, Cavendish Square, W. (C., 1896.)

1896 MACKINTOSH, ASHLEY W., M.A., M.D., Wellington House, Alford Place, Aberdeen.

1889 MACPHAIL, S. R., M.D., Borough Asylum, Rowditch, Derby.

O.M. MAGUIRE, ROBERT, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

,, MANN, J. DIXON, M.D., F.R.C.P., 16, St. John Street, Manchester.

1894 MARTIN, SIDNEY, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1893 MAUDE, ARTHUR, L.R.C.P., M.R.C.S., Westerham, Kent.

1891 MAY, W. PAGE, M.D., M.R.C.P., Helouan, Cairo, Egypt.

1890 MENZIES, WILLIAM FRANCIS, M.D., B.Sc., M.R.C.P., County Asylum, Rainhill, Lancs.

O.M. MERCIER, C., M.B., F.R.C.S., Flower House, Southend, Catford, S.E.

MEYER, ADOLF, M.D., Clark University, Worcester, Mass., U.S.A.

,, MICKLE, W. JULIUS, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (C., 1895-97.)

1895 MOORE, NORMAN, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. MORRIS, MALCOLM, F.R.C.S.Ed., 8, Harley Street, W.

1888 MOTT, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894, 1897.)

1894 MURRAY, GEORGE, M.B., M.R.C.P., 2, Saville Place, Newcastle-on-Tyne.

1889 MURRAY, H. MONTAGUE, M.D., F.R.C.P., 27, Savile Row, W.

O.M. NETTLESHIP, EDWARD, F.R.C.S., 5, Wimpole Street, W.

,, NICOLSON, DAVID, C.B., M.D., M.R.C.P.Ed., Elmhyrst, Guildford, Surrey.

1888 NIERMEYER, J. H. H., M.D., Amsterdam.

,, NORMAN, CONOLLY, F.R.C.P.I., F.R.C.S.I., Richmond Asylum, Dublin.

1887 OGILVIE, GEORGE, M.B., M.R.C.P., 22, Welbeck Street, W.

,, OGILVIE, LESLIE, M.B., M.R.C.P., 46, Welbeck Street, W.

,, OLIVER, THOMAS, M.D., F.R.C.P., 7, Elison Place, Newcastle-on-Tyne.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., The Bryn, Godalming, Surrey.

,, ORD, W. M., M.D., F.R.C.P., 37, Upper Brook Street, W. (C., 1893-94.)

,, ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

,, PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (C., 1891-2.)

1895 PARKINSON, JOHN PORTER, M.D.Lond., M.R.C.P., 40, Wimpole Street, W.

1896 PATERSON, DONALD ROSE, M.D.Edin., M.R.C.P., 18, Windsor Place, Cardiff.

1895 PINDER, GEORGE, M.B., B.C., Seafield House, Ramsey, Isle of Man.

1888 PITT, G. N., M.D., F.R.C.P., 24, St. Thomas's Street, S.E.

O.M. POORE, G. VIVIAN, M.D., F.R.C.P., 32, Wimpole Street, W. (C., 1898.)

1887 PRINGLE, J. J., M.B., F.R.C.P., 23, Lower Seymour Street, Portman Square, W.

O.M. PURVES, W. LAIDLAW, M.D., 20, Stratford Place, W.

1894 RANSOM, WILLIAM B., M.D., M.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 2, Harley Street, W.

O.M. REID, E. W., M.B., University College, Dundee.

1897 REISSMANN, CHARLES H., B.A., B.Sc., Saxony Villas, Oppidans Road, Primrose Hill.

1893 RENNIE, GEORGE E., M.D., 16, College Street, Sydney, N. S. Wales.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 23, St. John Street, Manchester.

1892 RICHARDS, JOSEPH PEEKE, M.R.C.S., 6, Freeland Road, Ealing, W.

1891 RIVERS, W. H. R., M.A., M.D., M.R.C.P., St. John's College, Cambridge. (C. 1897.)

1892 Rowe, Edmund Lewis, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1892 Ruffer, Marc Armand, M.D., Cairo.

1889 Russell, J. S. Risien, M.D., F.R.C.P., 4, Queen Anne Street, W. (Sec., 1896-1897.)

O.M. Sanderson, J. Burdon, M.D., F.R.C.P., F.R.S., Banbury Road, Oxford.

,, Savage, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres. 1897; V. P., 1895; C., 1886-90, 1898.)

1888 Savill, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

O.M. Schaefer, E. A., F.R.S., University College, Gower Street. (Pres., 1893; V.P., 1892; C., 1886-90.)

,, Semon, Sir Felix, M.D., F.R.C.P., 39, Wimpole Street, W.

,, Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (C., 1889-92.)

,, Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (C., 1892-95.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1895 Smith, F. J., M.D., M.R.C.P., 4, Christopher Street, Finsbury Square, E.C.

1887 Smith, R. Percy, M.D., F.R.C.P., Bethlem Royal Hospital, Lambeth.

1895 Smith, Telford, M.D., B.S., Royal Albert Asylum, Lancaster.

1895 Spicer, William T. H., M.D.Cantab., F.R.C.S., 47, Welbeck Street, W.

1895 Stansfield, Thos. E. K., M.B., Claybury Asylum, Woodford, Essex.

1895 Stanley, Douglas, M.D.Edin., 9, Easy Row, Birmingham.

1892 Starling, Ernest Henry, M.D., F.R.C.P., B.S., 8, Park Square, N.W.

1897 Stewart, Purves, M.A., M.D., M.R.C.P., National Hospital, Queen Square, W.C.

O.M. Stewart, Sir T. Grainger, M.D., F.R.C.P.Ed., 19, Charlotte Square, Edinburgh.

,, Stirling, Wm., M.D., D.Sc., Owens College, Manchester.

1897 Stoddart, Wm. H., M.B., B.S., M.R.C.P., County Asylum, Prestwich, Manchester.

1895 Stout, G. F., M.A., St. John's College, Cambridge.

1887 Suckling, C. W., M.D., M.R.C.P., 103, Newhall Street, Birmingham.

O.M. Sully, James, M.A., LL.D., 1, Portland Villas, Hampstead. (V.P., 1890-91.)

1889 Sutherland, Henry, M.D., M.R.C.P., 21, New Cavendish Street, Portland Place, W.

1888 Syers, Henry W., M.D., M.R.C.P., 4, Oxford and Cambridge Mansions, Hyde Park, W.

1891 Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W.

1898 Thomson, H. Campbell, M.D., M.R.C.P., 34, Queen Anne Street, W.

1889 Thorburn, Wm., F.R.C.S., The Limes, Nelson Street, Manchester.

1892 Titchener, E. B., B.A., Ph.D., 72, Heustis Street, Ithaca, N.Y.

O.M. Tooth, H. H., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896.)

1892 Trevelyan, E. F., M.D., B.Sc., M.R.C.P., 40, Park Square, Leeds.

1892 TUCKEY, CHARLES LLOYD, M.D., C.M., 88, Park Street, Grosvenor Square, W.

1892 TUKE, Sir J. BATTY, M.D., F.R.C.P.Ed., 20, Charlotte Sq., Edinburgh.

1891 TUKE, THOMAS SEYMOUR, M.B., M.R.C.S., Chiswick House, Chiswick.

1891 TURNER, WILLIAM ALDREN, M.D., F.R.C.P., 13, Queen Anne Street, W.

O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1888 VOORTHUIS, J. A., M.D., Medan, Deli, East Coast of Sumatra. (Communications to be addressed to M. Seyffardt, Bookseller, Amsterdam.)

1895 WADE, ARTHUR L., M.D., Somerset and Bath Asylum, Wells.

1894 WALKER, A. STODART, M.B., 30, Walker Street, Edinburgh.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 16, Grove End Road, N.W. (C. 1894-97.)

O.M. WARD, J., D.Sc., Trinity College, Cambridge.

,, WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1892 WASHBOURN, J. W., M.D., F.R.C.P., 6, Cavendish Place, W.

O.M. WATTEVILLE, A. de, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896; Sec., 1886-9; Editor of *Brain*.)

1897 WEBER, F. PARKES, M.D., M.R.C.P., 19, Harley Street, W.

1897 WEST, SAMUEL, M.D., F.R.C.P., 43, Wimpole Street, W.

1897 WHITE, ERNEST W., M.B., M.R.C.P., City of London Asylum, Dartford.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley Street, W. (C. 1897.)

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGGLESWORTH, JOSEPH, M.D., M.R.C.P., County Asylum, Rainhill, Lancashire.

1894 WILLIAMSON, RICHARD T., M.D., M.R.C.P., 294, Oxford Road, Manchester.

O.M. WILKS, Sir SAMUEL, Bart., M.D., F.R.S., 72, Grosvenor Street, W. (Pres., 1887; V.P., 1886; C., 1888-91.)

1896 WILSON, ALBERT, M.D.Edin., M.R.C.P., 33, Fairlop Road, Leytonstone.

,, WOOD, GUY M., M.B., M.R.C.P., County Asylum, Rainhill, Lancashire.

1889 WOOD, T. OUTTERSON, M.D., M.R.C.P., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., Examination Hall, Victoria Embankment.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., M.R.C.P., 37, Trevor Sq., S.W.